# THE ZULU DIABETIC

An account of the possible causes of diabetes in the Zulu people; the clinical findings when the patients were first, and subsequently seen; and the effects of treatment during, and the results of attempting, a nine-year follow-up in 133 Zulu Diabetic patients.

BY

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THESIS SUBMITTED FOR THE M.D., UNIVERSITY OF EDINBURGH.

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## DECLARATION

I, GEORGE DUNCAN CAMPBELL, declare that this thesis is the result of my own personal work between 1958 and 1968.

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## SUMMARY OF THE THESIS.

TITLE : THE ZULU DIABETIC.

SUB-TITLE: An account of the possible causes of diabetes in the Zulu people: the clinical findings when the patients were first, and subsequently seen: and the effects of treatment during and the results of attempting, a nine-year follow-up in 133 Zulu diabetic patients.

SUMMARY: This Thesis is an account of the study of 133 diabetics from the Nguni race (Zulu and allied tribes), and contains descriptions of attempts, (a), to set out as fully as possible all factors contributing to the emergence of diabetes in the Zulu people: (b), to follow for as long as reasonably possible (nine years) a representative group who appeared favourable subjects for such follow-up: (c), to find the incidence of the complications and concomitants of diabetes in a group of diabetics of varying duration of the disease at their first appearance at the Clinic: (d), by examining patients carefully at each 6-monthly follow-up, to study the emergence of diabetic complications or concomitants over and above those noted at the first visit: (e), to study effects of treatment and criteria of control in a long-term follow-up: (f), to carry out certain simple and complex clinical pathological studies on the present series: (g), to give an account of the difficulties encountered in the treatment and long-term follow-up of semi-literate patients: (h), to set out suggestions upon how best to deal with such diabetics, and (i), to include a section on the terms used in the Clinic, as a basis and guide for others seeking to make a simple language guide in other languages for the guidance of semi-literate patients.

Between 5th August, 1958 and the 5th February, 1959, 188 Bantu (chiefly Zulu) diabetics were registered at the newly-formed Diabetic Clinic of the King Edward VIII Hospital and the University of Natal. In order to eliminate patients attending Hospital for only short periods and who would thus not be suit-

able for long-term follow-up, only patients who attended the Clinic for a full year were included in the series. When the series was "closed" on the 5th February, 1959, there remained 133 of the original 188. These patients were followed for periods of up to nine years, and any still attending at the closure of the Study on 5th February, 1968 would have been followed for a minimum of nine years, though, as seen on p. 133 it is plain that many fell out long before this.

( Findings of the Thesis are summarised; ("Final) Summary of the Thesis", p. 145).

LIST OF CONTENTS.

PART III - THE CLINICAL STATE IN 133 ZULU DIABETICS. PAGE.

# APPENDICES.

## SECTION I - 1 INTRODUCTION.

Since the end of the Second World War in 1945, there have been rapid increases in the standards of living, employment and education in many communities that were previously underprivileged. Nowhere has this been more marked than in parts of Africa, and in no part has this change been so speedy as around the big cities of South Africa. Large numbers of people living in the country in tracts of over-grazed and poorly farmed land, known as the "Native Reserves", are still living a spare and pastoral existence. Those who have moved into the "Big Cities" - mostly Durban and Johannesburg - have achieved a strikingly better mode of life, principally as regards food intake, income and social and intellectual status.

This was by no means wholly true, as one realises during a visit to the former notorious slum suburb of Durban called "Cato Manor", the scene of the riots of 1959. These were known in the Zulu language as "Isidumo zamakosikazi", or "The War of the Ladies", and were popularised in the World press. They were triggered off by the destruction of illicit liquor stills, which were the source of so much income to many of the Zulu women, who were unable to support themselves as well in any other way, in an urban environment. Furthermore, the prevalence of deficiency diseases, such as Kwashiorkor, Beri-Beri and Pellagra, were still very markedly evident in these earlier suburbs. With the movement of the African people from slum suburbs to new housing schemes, the prevalence of many of these diseases dropped remarkably, and in particular it is interesting to relate that the Amoebiasis research wards have now difficulty in filling their beds because of the improved standards of hygiene in the new townships.

Evidence to support this advancement of standards is reflected in the large increase of circulation in the Zulu Weekly "Ilanga lase Natal" (The Natal Sun), over the last 22 years, amongst many other indications. These advances have been due to the rapid strides that have been made, mostly by their own exertions, by the Zulu people. Readers of even recent pictorial books about Natal would be sadly disillusioned by a visit to the Diabetic Clinic at King Edward VIII Hospital, where patients, instead of being scantily attired with a "row of beads", often come to the Clinic dressed

SECTION I - 1 (continued)

in the height of western fashion, though all are by no means literate. One of the most noticeable of the social advances of late, has been the remarkable rise in ownership by African people of their own motor cars, something which before the Second World War was almost unknown.

Increasing numbers of previously poor people now wear white collars and carry brief cases and indeed I have christened the disease, diabetes in the Zulu people, "Isifo Sikitasi" (The Disease of the Brief Case) and this is adequately borne out by the male patients attending the Clinic.

In this Thesis, the social and economic background of the parents of these diabetic patients has been compared with that of the patients themselves, and in almost every instance there has been a "great leap" forward in social standards with the passing of the last generation. All this social emergence, or "civilisation", was bound to bring in its train a change in the diseases to which the Zulu had previously been prone. This pattern is one which has been observed in many countries in the world, and has been the basis for a joint work on the effect of refined carbyhydrate on the human race, which was published in Britain in 1966. (Cleave, T.L. and Campbell, G.D. (1966) Diabetes, Coronary Thrombosis and The Saccharine Disease. J. Wright & Son, Bristol).

It was indeed a salutory experience for one who had practised medicine amongst Zulu people, to have gone to America to work in a hospital there. I was greatly surprised by the large numbers of American negroes who were suffering from myocardial infarction, peptic ulcer, benign and malignant hypertension, thyroid disease and particularly from diabetes - conditions, two decades ago, that were seen most infrequently in the Zulu people. A case of peptic ulceration in a Zulu patient would, until recently, have aroused in the Wards of the King Edward VIII Hospital in Durban, as much discussion as would have a case of amoebic hepatitis in the wards of a Scottish hospital.

Over the last 20 years, therefore, the scene has changed in our medical wards. Previously the vast majority of our patients were suffering from the infective and deficiency diseases; now, though

SECTION I - 1 (continued)

the majority still suffer from the same conditions, we find in the African wards many cases of benign hypertension in obese patients, occasional patients with myocardial infarction, patients with cerebral vascular accidents and diabetes, and on one occasion recently, in a medical charge of 50 beds, there lay no less than three cases of peptic ulcer.

In view of the apparent commonness of diabetes in the American negro, it appeared that diabetes would inevitably be found in increasing numbers amongst the Zulu people with their social advancement. This is certainly true amongst the town dwellers. However, it continues to be a most uncommon condition in the country. The present series initially collected up to February, 1959, though small by overseas standards, was very large in the eyes of many Natal doctors, most of whom have never seen a Zulu diabetic in many years of practice. However, at the present time the number of Zulu patients who are registered at our Clinic, is 2,106: the rate of registration has quickened in the last few years, and is notably formed by large numbers of very fat middle-aged patients. These now greatly outnumber the Insulin-dependent patients, which before, as in this series formed 1/4 of the patients but have now dropped to about 1/20th.

In addition, I have shown how the aetiological studies of diabetes in the Zulu are greatly facilitated by the fact that there are two distinct populations - the settled urban Zulu who is liable to develop diabetes, and the settled country patient who is very uncommonly diabetic. Between these two groups are marked cultural, economic and dietary differences, and the "emergence" of diabetes in people who have moved from the country into the town, is mirrored in the fact that diabetes and good nutrition go together. Indeed I have referred to diabetes as an "Illness of Well-Being" (Campbell, G.D. (1963) East African Medical Journal 5, 272). In fact, in the Zulu in Natal, under-nutrition and diabetes are mutually exclusive, with the exception of the uncommon porphyria - diabetes-cytosiderosis syndrome. Furthermore, it was of great interest to myself to have visited Diabetic and Endocrine Clinics in Germany, Scandinavia, Russia and Greece in 1960, to see

SECTION I - 1 (continued)

how the diet of the peasant Zulu patient approximated so closely to that eaten by European people in the occupied countries during the 1939 - 45 war, when the incidence of diabetes fell markedly: whereas the diet of the settled urban Zulu is very similar to that seen in Europe during the economic recovery of the late 1940's and early 1950's, with particular reference to the enormous increase in sugar intake as compared with the war years, and to a lesser extent increased intake of protein and fat, at the same time as the incidence of diabetes began to rise towards pre-war levels.
Therefore, it is against this background that it can be seen that the political effects of the "winds of change" in the African Continent have been antedated by about 10 years by remarkable changes in the pattern of clinical medicine - particularly in those living in the larger towns, who have become rapidly socially advanced.

The history of diabetes in the Zulu people has only been passed down in lore, and we are indebted to the writing of the Rev. Arthur Bryant, an English clergyman mentioned in the dietary predictions later on in this Thesis, who has drawn attention in his book "Zulu Medicine and Medicine Man" to a disease called "Umxhoboko" when he first wrote in 1907. (Bryant, A.T. Published 1967. Zulu Medicine and Medicine Man - C. Struik, Cape Town, p. 26).

This disease, "Umxhoboko", was classically referred to as the "King's Disease", as it first afflicted the King Mpande. It was described as a disease "which afflicted fat people and yet wasted their bodies from the inside". It is notable that with the passing of the great militant Kings, Chaka and Dingaan, their stay-at-home brother, Mpande, who was so obese that he could not walk and had to be dragged about on ox-skins or a small cart, became King of the Zulu nation, and he died from this "King's Illness". The Rev. Bryant was very interesting in his observations that this disease was associated often with the presence of tuberculous glands and fistulae in the neck, and it is quite remarkable that this keen observer should have wondered why this disease, (in other words, tuberculous neck abscesses) should be seen more particularly only in the very poor, or the enormously rich overweight patients; this is almost

SECTION I - 1 (continued)

certainly due to the fact that in the instance of Mpande, the diabetes had precipitated the tuberculous infection in the neck, whereas in the poorer people tuberculosis was particularly rife.

Though in this Thesis clinical descriptions are primarily about the patients included in the series itself, where clinical findings of particular interest have been seen in Zulu diabetics not in the series, short accounts of these have been included in the appropriate section.

SECTION I - 2 REFERENCES TO PREVIOUS LITERATURE.

Up until the Diabetic Conference at Dar-es-Salaam in 1963, the references to African diabetics were particularly sparse in the medical literature. With the exception of a short account of 17 cases, (Gelfand, M. (1957) The Sick African, Juta & Co., p.696 ) and a more recent paper (Reef, H.E., Seftel, H.C., and Kaplan, Y.S. (1960) Medical Proceedings, 6. 13), there have been few studies reported with the exception of papers by myself (Summarised in (Campbell, G.D., (1963) S.A. Med. J., 37, 48, 1195). In Gelfand's study in Salisbury he said he had been able to collect 17 African diabetics in 2 years and he stressed the rarity of the condition, and the great difficulty of treating the African diabetic. In fact he says in his differential diagnosis of coma in the African patients, that "hypoglycaemic coma does not come into the diagnosis" since the African is not likely to be receiving Insulin outside the hospital. Fourteen of his 17 patients were male , and the age of onset was most commonly under 40 years. In the other study mentioned above, there was a brief account of a series of 113 mixed Bantu patients which were collected over exactly the same length of time, that the King Edward VII Diabetic Clinic took to collect 250 patients. In spite of the fact that their Clinic is small and that their hospital drains an enormous population, probably twice as large as that drained by the King Edward VIII Hospital, they regard diabetes in the African as a "common and important" clinical problem. The present day importance of this clinical problem is now well borne out by my present total series of over 2,000 African diabetics. 75% of Reef's patients were over 40 years of age and only 3% under the

VIII

SECTION I - 2 (continued)

age of 20 years. The sex distribution shows the 3 : 2 predominance of females : two-thirds of the patients are overweight. Surprisingly, they add two statements - "In view of our success with dietetic therapy, we have not used the oral anti-diabetic agents" and "three-quarters of our patients are on Insulin"; interesting, when one remembers that two thirds of their patients are overweight.

However, in 1963 (November 30th) there appeared an important issue of the South African Medical Journal, Volume 37, No. 48, pp 1193 et seq. This issue was totally devoted to the subject of diabetes in non-white people in Southern Africa, and in it was summarised all published work, and in the reference lists was mention of every paper that had been written on this particular subject. In this issue, to which particular reference is made here, were extensive papers from Durban, Johannesburg, Salisbury (Rhodesia) and Cape Town. In particular, papers with considerable information about Bantu (African) diabetics were :-

Campbell, G.D. (1963) Diabetes in Asians and Africans in and around Durban. ibid. 1195.

Gelfand, M., and Forbes, J.I. (1963) Diabetes Mellitus in the Rhodesian African, ibid. 1208.

Seftel, H.C., Keeley, K.J., and Walker, A.R.P. (1963) Studies in Glycosuria in Non-White Populations of the Transvaal, PART ONE. ibid. 1213.

Jackson, W.P.U., (1963) Diabetes in the Cape Peninsula, ibid 1220.

The most important publication to which comparative reference should be made is the monograph :-

Tulloch, J.A. (1962) Diabetes Mellitus in the Tropics. E & S. Livingstone (Edin. & London). Here very ample earlier reference has been made to work done in Southern Africa in Clinics catering for Bantu (African) diabetics and compared with observations elsewhere.

## SECTION I - 3 THE HOSPITAL AND CLINIC BACKGROUND.

The King Edward VIII Hospital in Durban is an institution of close on 2,000 beds, and is the chief non-European hospital in the Province of Natal, and one of the largest hospitals in the Southern Hemisphere. It is situated near the inner harbour and is near the more industrialised parts of the town. It is the hospital upon which the Medical School of the University of Natal is based. The Hospital records annually 700,000 out-patient attendances, of whom about one-tenth are admitted. 80% of this total are Zulu patients and the rest are Natal Indians. Up until 1958 a free service was given to Zulu patients, but in order to cut down on the seething masses attending, a charge of 40 cents (4/-) per patient was levied at that time, to those adjudged able to afford it. This had the effect of cutting attendances down about one-sixth in the year after its imposition, but soon after this the attendances shot up again. In 1967, alarmed at the massive financial loss of the Medical Services, the Provincial authorities have not instituted a system whereby the better-off patients are charged R1.90 cents, the poorer ones 40 cents per patient, but the indigent ones are now still treated without charge.

The name of the Hospital in Zulu is "Kwa Khangela" (Anglice-Congella), and means an 'observation post' as the Hospital is situated on a place where there used to be a look-out post set against certain marauding and cannibal tribes. The Hospital has a fine reputation amongst the Bantu people, large numbers of whom will by-pass the hundred or so hospitals scattered about the Province in order to be treated at a place that they regard as the Mecca of Therapeutics. As the out-patient "sieve" has a very fine mesh, only patients requiring energetic or special treatment are admitted, and as the admitted patients are often suffering from serious and advanced disease, the study of their pathology is a never ending succession of fascinating gross morbid anatomy. Few Departments are able to do justification to the veritable mine of clinical material that is available. In certain diseases, such as amoebic hepatitis, porphyria, liver

SECTION I - 3 ( continued)

carcinoma and tuberculous meningitis for instance, there are at any time in the wards of a single hospital more cases than will be described in a whole year in Great Britain.

The establishment of a Diabetic Clinic in August 1958 drew further attention, not only to the enormous numbers of Natal Indian diabetics, but also to the fact that diabetes in the Zulu was either becoming more common, or was beginning to be diagnosed. The most impelling cause for clinic attendance is the prohibitive cost of anti-diabetic drugs, particularly the oral substances.

The Clinic registers no less than 22 new diabetics weekly, continuously, mostly Indians, and the numbers do not appear to be slacking off. In just over 9 years of existence, we have 10,500 patients on our books, and an average attendance of about 350 per week; of these, one-fifth are Zulus. The 133 Zulu diabetics, comprising the present series, have now all been followed for 9 years, during which the absolute default rate has been high and is summarised in the following table :-

133 ZULU DIABETICS

SUMMARY OF DEFAULTERS, DEATHS AND PATIENTS STILL ATTENDING AT THE END OF CONSECUTIVE YEARS. (a)

(all percentages to 1 significant figure).

| YEARS | 1 | -2 | -3 | -4 | -5 | -6 | -7 | -8 | -9 present time | Totals |
|---|---|---|---|---|---|---|---|---|---|---|
| STILL ATTENDING (% age) | 133 | 84 | 69 | 55 | 47 | 39 | 33 | 32 | 31 | 31 |
| | 100% | 63.3 % | 51.9 % | 41.4 % | 35.4 % | 29.4 % | 24.8 % | 24.1 % | 23.4 % | 23.4 % |
| In each year:- DIED (known to have died) | - | 5 | - | - | - | - | - | - | 1 | 6 |
| | | 3.8 % | | | | | | | 0.8 % | 4.5 % |
| DISAPPEARED | - | 44 | 15 | 14 | 8 | 8 | 6 | 1 | 0 | 96 |
| | | 33.1 % | 11.3 % | 10.5 % | 6.0 % | 6.0 % | 4.5 % | 0.8 % | 0.0 % | 72.2 % |

(a) - see over.

SECTION I - 3 (continued)

(a) All patients had to attend for a full year to qualify for this series.

From the outset, in running the Clinic for Zulu patients, two objectives have been paramount : (1) That no patient should be admitted to Hospital unless strictly necessary, because of perennial serious shortage of beds in the Hospital, and (2) that to save adding more work to a grossly overworked Laboratory service, patients should be assessed by the clinical criteria that have been set out on p.122 The second one is fairly easily justified. Though attitudes have changed over the 9 year follow-up, in 1958 many Zulus, especially the men, believed that to have blood taken is to weaken one. In a series of patients suffering from hepatic cytosiderosis and diabetes (now believed to be indistinguishable from haemochromatosis), attempts to treat patients by means of venesection, inevitably stopped after the first pint was seen to be withdrawn, until arrangements were made for the patient to be in one half of a room divided by screens, and his arm alone being in the other room, where venesection was done under local anaesthetic, so that he could not see what was going on. Even this did not have much success after an initial period.

SECTION I - 4 RACIAL AND ETHNOGRAPHIC BACKGROUND.

In order that there should be no confusion about racial nomenclature, I would like to define the terms "Bantu", "Nguni" and "Zulu".
BANTU: In modern English usage this term refers to any Southern African of negro extraction. The word itself in the classical Zulu means any 'people' ("Abantu"). Thus "Abantu abanhlope" literally means "White people"; "Abantu anpofu" means "Yellow people - the Cape Coloureds: and "Abantu Abansundu" (or "Abantu Abanyama") means "Brown People", or, in fact the people who are commonly referred to as "Bantu". The term "Bantu" is used synonymously with the term "African" - used by many to refer to negroes from Africa. To avoid confusion in this Thesis, the people are referred to as "Bantu (African)".

SECTION I - 4 (continued)

NGUNI: The Nguni Race are the descendants of the large southward migration of Bantu to the East of the Drakensberg range, which took place at about the time that Diaz was rounding the Southernmost tip of Africa at the end of the fifteenth century. They comprise the ethnic group that is studied in this Thesis, and contain the following tribes from North Southwards:-
The Zulu, The Maci, The Bacas, The Pondos, The Xosas and the Fingoes. The majority of my patients were pure Zulus, and particular care has been taken to exclude patients who may have been admixed with European blood. It is interesting to note that the warlike Matabele who inhabit most of Southern Rhodesia, are members of the Nguni race, who embarked upon a retrograde migration as a result of the possibility of their being included in one of the many blood baths indulged in by the legendary Caesar of the Zulus - Chaka.

ZULU: This word is derived from the name of the biggest and most warlike of the Nguni tribe, namely the "Amazulu" or the "inhabitants of Heaven". The name is that used by the Royal Family, and the actual bearers of the name are entitled to the Royal Saulte "Bayete!" The actual origin of the name was that of one of the Chieftains of an earlier faction of the Zulus. Original Chieftains were two :- Zulu and Dhlamini. There is a good deal of disagreement about this, and I do not intend to enter here into a discussion of this. (For a full account:- Bryant, A.T. (publ. 1964) History of the Zulu and Neighbouring Tribes, C. Struik, C.Town (Part Eight), 125).

SECTION I - 5 COMPOSITION OF THE SERIES.

Between 8th August 1958 and 5 th February 1959, 188 African (Bantu) diabetics were registered in the newly formed Diabetic Clinic of the King Edward VIII Hospital in Durban (Nos. A/1 - A/188). In order that a sound basis might be formed for long-term follow-up of a certain number of these patients and to avoid including those who attended for perhaps a single visit only, attempts were made to follow this whole series of patients for at least one year, that is

SECTION I - 5 (continued)

to say until the 5th February, 1960. At the end of that time all non-Nguni (mostly non-Zulus) were excluded from the study, plus those who had defaulted during the year, or for other reasons were considered unsuitable for long-term follow-up. This left 133 diabetics (See Appendix One - for Clinic reference numbers of patients included), and the results of the attempted one-year probationary follow-up are set out in the following table :-

188 BANTU (AFRICAN) DIABETICS.

| | | |
|---|---|---|
| NGUNI RACE | ZULUS ..... | 127 |
| | XOSAS ..... | 4 |
| | BACA ..... | 2 |
| Present Series ..... | | 133 |
| OTHER BANTU | SESUTO..... | 5 |
| | SECHUANA .. | 1 |
| NOT FOLLOWED LONG ENOUGH... | | 45 |
| INFORMATION NOT ADEQUATE... | | 4 |
| | | 55 |
| TOTAL OF BANTU DIABETICS..... | | 188 |
| TOTAL OF INDIAN DIABETICS.... (collected during the same period) | | 731 |
| TOTAL ATTENDERS AT THE KING EDWARD DIABETIC CLINIC COLLECTED OVER THE TIME OF COLLECTION OF THE SERIES. | | 919 |

The proportion of Zulu to Indian diabetics, namely 1 : 4, has remained remarkably constant over the nine years in review. The prime intention was to study clinical features as seen to exist in a group of diabetics of 0 - 12 years' duration, when they were first seen, and to try and establish the proportion of patients suffering from diabetic complications or concomitants. Then it was intended to follow the 133 patients by examining them regularly to try and give some indication of the frequency, and after what duration of diabetes, that these complications were seen to emerge.

SECTION 1 - 6 METHODS OF DIAGNOSIS IN 133 ZULU DIABETICS.

| | Males(53) | Females(80). |
|---|---|---|
| Clinical and Blood Sugar Investigations. | 43 | 66 |
| Clinical and Blood Sugar Investigations diagnosed as above and referred from other Hospitals | 6 | 9 |
| Clinical and Urine tests only (Case 21 diagnosed his diabetes by tasteing his urine) | 4 | 5 |

Many of our patients were referred to the Clinic after an initial period of stabilisation in the Wards of our own Hospital,, but many new diabetics were referred directly from the Medical Out-Patients Department. In addition, there have been a number of patients who have been diagnosed at other hospitals, principally from McCords Zulu Hospital, and subsequently referred to our Clinic. It will be seen that only 10 of this series were diagnosed on clinical grounds and urine testing alone. I have yet to see a case of renal glycosuria in the Zulu, and I have felt justified in regarding as diabetic, patients who have presented with a heavy glycosuria, with the typical complaints of polyuria, polydipsia, polyphagia, pruritus pudendi and loss of weight, all or any being present, especially if they have subsequently responded to dietary measures and anti-diabetic therapy.

SECTION I - 7 DURATION OF DIABETES WHEN PATIENTS WERE FIRST REFERRED.

| | Males | Females |
|---|---|---|
| New Cases (referred upon diagnosis | 18 | 44 |
| 0 - 5 Years (referred after an initial period of treatment) | 23 | 25 |
| 5 - 10 Years | 8 | 9 |
| Over 10 Years | 4 | 2 |
| TOTAL ..... | 53 | 80 |

SECTION I - 7 (continued)

Nearly half of these were new patients, and most were seen before their diabetes had lasted for ten years. Only six patients had had the disease for over ten years, in marked contradistinction to the Indians at the same Clinic, many of whom were very long-standing diabetics. "Duration" in this context refers to how long the patient has been aware of diabetic symptoms, or in the occasional asymptomatic case, to how long he has been aware of being diabetic.

## SECTION I - 8. THE GROUPS OF DIABETICS IN THE DIABETIC CLINIC AT KING EDWARD VIII HOSPITAL.

Though we now employ a simplified nomenclature in the Clinic, as a matter of historical record, the following table is set out to show the terms previously used in the Clinic in respect of the Zulu diabetics. For obvious reasons the terms "Type One" and "Type Two" were discarded, for though they have been used frequently to categorise diabetics in certain Clinics, they hold no cognisance of the important and discrete group of Senile Diabetics, who differ from the middle-aged patients, in that they may be truly Insulin-deficient patients. The terms previously are used in the Clinic as follows :-

Senile, Middle-aged, Young Insulin-dependent, "J" type ( and chronic pancreatitis) patients. Each of these groups is discussed in detail in Sections III 2 - 6, but the table is inserted here as the terms are used frequently in the discussion of aetiology of diabetes in the Zulu race. A further group, known as the "Insulin-independent young Diabetic" was described in the Natal Indian from the King Edward VIII Hospital in 1960. (Campbell, G.D. (1960) Brit. Med. J., 537). Only two of the present series of patients can be said to come into this group, which is discussed in more detail on p.56. . In the following Table these two cases have been included in the category of Insulin-dependant young diabetics. The following table which was made up before the description of this group these two cases were included in the category of Insulin-dependant young diabetics, as at that time they were actually on Insulin.

SECTION I - 8 (continued)

In spite of the very high incidence of Cytosiderosis in the Bantu, a clinical state that is hard to differentiate histologically from Haemochromatosis in the European, the syndrome of siderotic diabetes appeared in 1959 to be very uncommon in the Zulu, but subsequently a number of cases have been described from Johannesburg. (Seftel, H.C., Keeley, K.J. and Walker, A.R.P. (1963) S.A. Med. J., 39, 48, 1214). Certainly no case in the present series was proven as having pancreatitis, and a series of four post-mortems performed on diabetic Zulus in 1957, failed to show any evidence of pancreatic fibrosis. Chronic pancreatitis, itself, is very rare in the Bantu and none of the present series complained of any symptoms that might have been caused through a pancreatic steatorrhoea. Gelfand, M. (1957, The Sick African, Juta & Co., C.Town, p. 696) noted that pancreatic disease was rare in the African, and that none of his 17 diabetics was shown to have it. Higginson, J. (1953), (Am.J.Path, 29, 779) noted that siderosis was present in the pancreas of only 7 out of 32 heavily siderotic patients. Pancreatic fibrosis was said to be "rare" in the Bantu siderotic, and diabetes was also noted to be "rare" in the siderotic.

The abdomens of 1,254 consecutive non-white diabetics were X-rayed (Postero-anterior views) in the Diabetic Clinic of the King Edward VIII Hospital. Of these 293 were Zulu diabetics, and only one Zulu patient showed pancreatic calcification (case not in this series). This gives some idea of the possible importance of this condition in the Zulu diabetic.

SECTION I - 8 (continued)

GROUPS OF DIABETICS (KING EDWARD CLINIC NOMENCLATURE)
UP TILL 1960.

| | MALES | FEMALES | TOTAL |
|---|---|---|---|
| (A) SENILE DIABETICS. Generally over 60 yrs. at onset; mostly easy to control with small dose of Insulin or Tolbutamide. Mostly thin, and Zulus who have not lived in the towns - "Peasant Diabetics". | 5 | 8 | 13 |
| (B) MIDDLE-AGED DIABETICS. Most over 40 years at onset; mostly overweight; dietary control poor, but most are "satisfactorily" controlled on Tolbutamide, or Phenformin. | 32 | 56 | 88 |
| (C) YOUNG (INSULIN-DEPENDENT) DIABETICS. Under 40 years at onset; mostly thin; control often remarkably good on Insulin. Liable to ketosis if Insulin withdrawn. | 16 | 16 | 32 |
| (D) "J" TYPE DIABETICS. Under 40 yrs. at onset; thin, liable to Ketosis if Insulin is withdrawn. (8% of Cosnett's series of Natal Indians; 2% of Campbell and McNeill's series of Natal Indians). This variant is now believed not to exist. | NIL | NIL | NIL |
| (E) CHRONIC PANCREATITIS PATIENTS. | NIL | NIL | NIL |
| TOTALS ..... | 53 | 80 | 133 |

SECTION I - 8 (continued)

One can see that most of this series fell into the category of middle-aged diabetics, most of whom are or have been overweight. Ten percent of the patients were senile diabetics, and about one quarter were young Insulin-dependant diabetics, who, (similar to European diabetics of the same age), are sensitive to Insulin and lapse into ketosis and coma when their Insulin is withdrawn. In the present series there was no case of chronic pancreatitis.

During the last four years we have used a simplified form of nomenclature in an attempt to do away with code names and in an attempt to describe exactly the clinical condition of each patient. Simply, as regards age, patients are divided into young, middle-aged and senile, and as regards treatment, are described as being "Insulin-dependant" or "Insulin-independant". Thus, we have no "types" and rely upon the use of these six descriptive titles if we want to assign a patient to any particular group.

SECTION I - 9 INCIDENCE OF THE DISEASE.

Using the word "incidence" in the strict sense, it is quite impossible to assess any disease from the hospital populations, and until recently no actual house-to-house incidence studies have been done on the Bantu African people. There are large numbers of factors which influence the attendance of patients at any Clinic, and it is probably even not valid to try and compare the incidence of diabetes in racial groups by the proportion of their attendances at any one hospital. Attendances are limited in rural people by travel, and in urban people often by those who are well off enough to have private doctors; they would rather attend privately than attend hospitals. If one regards diabetes as an "illness of well-being", it would be expected that this sort of person would tend more to avoid our Clinic. There are a number of studies on hospital populations that have been published, and they are so diverse that to quote them would be misleading. On the grounds of earlier studies it is only possible to say that diabetes would appear to be inordinately rare in the rural population, but becoming fairly

SECTION I - 9 (continued)

common in urban dwellers.

Lately, I and Dr. Ribiero, a General Practitioner, combined to do a house-to-house Glucose Tolerance test survey of two thousand Bantu (African) people over the age of 10 years, in a township outside Pretoria which had 1/3 of its population Zulu, 1/3 Basutos and 1/3 other Bantu people. The first part of this study has been completed and this consisted of taking a two-hour blood glucose figure after a load of 50 gm. of glucose, and this has shown an overall incidence (in a house-to-house survey according to our borderline level of 120 mgs. at 2 hours using the auto-analyser with a potassium ferricyanide principle,) of 2.0% of the general population being above this figure. It is interesting to note that in this survey there was 100% acceptance in the population concerned.

Studies on diabetes incidence from the whole continent of Africa are extremely scanty and mostly invalid, and the whole subject was summarised in a panel discussion of the 1967 Diabetes Congress in Stockholm by myself under the title "The Incidence of Diabetes in Africa" (in print).

SECTION I - 10. MORTALITY OF DIABETES IN THE ZULU.

It is quite impossible to present any information about the mortality of this disease in the Zulu people, as even death certificates in civilised or advanced countries have been shown to be valueless in assessing diabetes mortality (Diabetes Mellitus - Ed. L.J.P. Duncan (1965). Pfizer Medical Monograph No. 1, 145): in a country where the certification of death in Bantu African people is only of recent introduction, it is quite valueless to try and come to any conclusion. In this present series of 133 people followed for nine years, at the end of this time, only 31 patients were still attending; 6 are known have died. Thus causes of mortality cannot be set out with certainty. However, when one studies clinic notes of the last visits of patients before they disappeared, certain conditions would appear to have been strikingly commonly described in such visits - namely acute tuberculosis, sudden hypertension and albuminuria, sudden heavy albuminuria

SECTION I - 10 (continued)

without hypertension, and, in particular, periods of very poor control in certain patients whose standards of looking after themselves was not satisfactory.

## SECTION II THE AETIOLOGY OF DIABETES IN THE ZULUS.

### Section II - 1 The three dietary eras in the development of the Zulu people in the last 100 years.

My work over the last 10 years has satisfied me that the syndrome of diabetes in the Zulu people is one in which the environmental factors have swamped those due to heredity. This is borne out by the probable absence of the disease three or four decades ago, the absence of family histories in the vast majority of cases of Zulus, and the absence of the disease in people of identical stock to those prone to diabetes, who have lived a peasant existence rather than moving into town.

Thus, in the aetiology of the disease in this context, thoughts turn at once to (a) dietary differences between urban and rural Zulus, (b) differences seen in the Zulu people as a whole over the last 100 years, and (c) differences in diet between Zulus and other ethnic groups.

Thus, the dietary changes that have occurred in these people have been fairly distinct, and there are three "eras" which I now intend to describe :-

(1) The animal product era - up till the 1895, the Rinderpest of cattle.
(2) The maize or cereal era - from 1895 to 1945.
(3) The refined carbohydrate era - 1945 onwards.

(1) The Animal Product Era.

With the exception of the last 70 years, for all their existence in Southern Africa, the Zulus have been a pastoral and nomadic people. This period lasted up until 1895, when their herds of cattle were wiped out by the disastrous Rinderpest ("Ukufa kwezinkomo" or more simply "Ulindapese"). Thus the Zulu people were turned in the space of a few months, from eating meat and drinking milk, into cereal eaters.

Though the cattle of the Zulu people were finally wiped out by the Rinderpest, it is of interest to note that there were earlier factors which contributed to a gradual decline in the numbers and health of the cattle, which had taken place for something

SECTION II - 1 (continued)

like 20 years before the Rinderpest. Cattle and wealth in the Zulu's eyes are synonymous, and at the coronation of the King Cetshewayo in 1873, he ordered that all his 100,000 beasts should be gathered in the Coronation Kraal at Ulundi and paraded past him. Not only did this last for some days, but the already tired beasts were faced with the long journey home. Some of the cattle at the march past were infected with bovine lung sickness (a virus condition) which spread through many of the herds at that time, and which was subsequently disseminated to all corners of his kingdom after the coronation.

(2) The Maize or Cereal Era.

Following the disastrous Rinderpest, when the Zulus turned, through necessity, almost overnight to agricultural methods of sustenance, the pattern of disease amongst them was characterised by the emergence of certain conditions due to unsupplemented cereal diet, and amongst these were the cytosideroses and the deficiency diseases, of which pellagra and beri-beri were the most important. It would appear in this era, when the general resistance of the people was lowered by this change from animal to inferior plant protein, that the infective disorders introduced by the white and Indian people, particularly tuberculosis, took an awful toll of the Zulu people. It was in 1907 that a Church of England clergyman, the Rev. Arthur Bryant,(x) predicted that the change from pastoral to agricultural farming would have a profound affect in weakening the Zulu nation in many ways, especially in their health. Thus the maize or cereal era may be said to last from 1895 to a period just after the Second World War, when the urban dwellers then entered into another era, but rural dwellers have continued as in the maize or cereal era, though rural circumstances have changed recently with the enormous efforts to distribute sugar in the rural Zulu areas. Thus, there has been a rapid increase in per capita annual consumption of sugar, even in the humblest peasants.

(x) (Bryant A.T. (1907). Pamphlet prepared for the Government of Natal Archives, Natal Museum, Pietermaritzburg. pp 1 - 9)

SECTION II - 1 (continued)

(3) The refined carbohydrate era.

This era has lasted, roughly, for the last 22 years. During this time there have been radical and sudden changes in the diets of the Zulus living in the towns, and the chief of these has been a change from the ingestion of the unrefined carbohydrates, such as home-prepared cereals, to (a) white bread of the highest degree of refinement, (b) sugar and farinaceous foods, and in particular (c) by a growing and now massive ingestion of sweetened soft drinks. It is during this era that there has been a rapid emergence of gross obesity (exceeding that seen in North America - Slome, C., Gampel, B., Abrahamson, J.H., and Scotch, N. (1960) S.Afr. Med. J., 34, 505), diabetes and hypertension. During the refined carbohydrate era, the rural dwellers have not been thus so exposed to this drastic change in diet. They have continued, until recently, largely to subsist on the foods of the cereal or maize era, and they still suffer from diseases characterising that era, having as yet not started to develop those mentioned above, which have emerged subsequent to the change towards highly refined carbohydrate foods.

It is against this background, that the emergence of the "Disease of Civilisation" must be viewed in the Zulu urban dweller, and in particular the difference in disease spectrum between the rural (unrefined carbohydrate eaters) and the urban dweller (refined carbohydrate eaters) must be emphasised. This important difference has been the basis of epidemiological studies that have been done comparing the rural and urban Zulus, as well as Indians in Natal, and Indians in the areas of great privation in India from whence they came to Natal about 100 years ago.

## SECTION II - 2 THE PREDOMINANCE OF URBAN DWELLERS IN THE PRESENT SERIES.

In handling diabetics, one is struck at once by the preponderance of town dwellers, as the following table shows :-

BREAKDOWN OF PATIENTS BY DIABETIC GROUPS INTO TOWN AND COUNTRY DWELLERS.
(i.e. more than four years in the big cities)
IN 133 ZULU DIABETICS.

| DIABETIC TYPE | TOWN DWELLERS | COUNTRY DWELLERS. |
|---|---|---|
| SENILE DIABETICS | NIL | 13 (all peasants except one) |
| MIDDLE AGED DIABETICS | 80 | 8 (all these patients had had a far more liberal dietary intake than the peasant diet) |
| YOUNG DIABETICS | 19 | 13 |
| TOTAL ... ... ... | 99 | 34 |

The predominance of urban dwellers amongst diabetics is borne out by McNeill, formerly the Superintendent of the 400-bed Eshowe Hospital, the largest general hospital in Zululand, which drains three or four hundred thousand patients. (McNeill, W.G., 1960 - personal communication). In his three years as Superintendent he saw only nine diabetics. He feels, in common with myself, that the low incidence of diabetes in Zululand itself is due to the fact that he deals "with a predominantly rural population, which is as yet little influenced by European dietary habits". The above table is interesting, in that the senile diabetic is exclusively a country dweller (hence an earlier term "peasant diabetic). The middle aged diabetic is almost exclusively a town dweller, and a glance at the very high carbohydrate diet of the urban Zulu will indicate why: Insulin-dependant patients come almost equally from both regions.

## SECTION II - 3 COMPARISON OF DIETARY AND OTHER HABITS OF COUNTRY PEASANTS AND TOWN DWELLERS.

Amongst the Zulu people we have, therefore, as regards the risk of developing diabetes, two fairly well defined groups: firstly the town dweller, who appears to be more particularly liable to develop diabetes, and the other "diseases of civilisation": and, secondly, the country peasants, who are far less liable to develop diabetes if they are not influenced by European habits, unless they come into the category of "senile" diabetics. From the first, attempts have been directed in trying to find whether there are any significant differences in the dietary habits of these two groups. Dietary intake has so much bearing in the incidence of other diseases in the Zulu (notably cytosiderosis and porphyria), that it would be interesting to note any differences, particularly in the ingestion of carbohydrates (and particularly of sugar): protein apparently spares the liver of the Bantu against siderosis, and in view of the fact that in a small series of liver biopsies on the Zulu diabetic showed only one patient with minimal siderosis, it would be most pertinent to review the protein intakes of the country peasants, who are particularly liable to siderosis, and the town dwellers who are not. In this respect the diets of the present series of diabetics have been compared with those of a number of severely siderotic patients. It has also been interesting to review the diets of the present series, in the light of that of their parents' diets. In almost every case there has been a "great leap" forward in their social and financial status over the last generation.

If diabetes can be referred to as "an illness of well-being", then it is natural to look to those Zulu families who have been in a fortunate position economically over some generations. The classical example is the Royal Family. The emergence of the disease in this family probably coincided with the passing of the superbly fit front-line militant kings, such as Chaka and Dingaan, and with the accession of their slothful and stay-at-home brother, Mpande, who was so fat that he couldn't walk, and had to be dragged about on ox-skins, or in a small cart. He died from the "King's Illness" (Umxhobokho), which is described

SECTION II - 3 (continued)

as being an illness "which afflicts those that are fat, and yet melts their bodies and muscles from the inside". Since then diabetes, as it almost certainly was, has assumed epidemic proportions in both direct and cadet lines, aided by marriage with other related and noble families which also contain diabetics.

In our first 150 Zulu diabetics, the surname "Dhlamini" occurred with such frequency (without family relationship) as to stimulate enquiry as to whether this appearance was significant. The percentage of Dhlamini's in our diabetics was about 10 times that seen in a non-diabetic control group of 200 Zulu out-patients attending the Medical Out-Patient Department for various reasons. The name is made of two words "Idla emini" - denoting a man who eats heavily in the middle of the day, as well as at his main meal at night!

Our study of dietary aetiology in the Zulu diabetic is, therefore, greatly facilitated by two things (1) the virtual absence of diabetes in the rural areas, and (2) the very great differences between the diet of rural and urban Zulus.

There are two superb dietary surveys that were made during the 1950's amongst the Zulu people. The first was a very careful survey that has been made by the Institute of Family Health and Community Planning of the University of Natal, on the dietary intakes of a large number of Zulus who are living in the Lamontville Location, just to the South of the City of Durban. (Gampel, B. (1959) personal communication). These people are typical of those referred to in this thesis as "urban" or "town dwellers". To live in this particular location fairly stringent rules apply. For instance, all people there must be legally married. All the men and most of the women are gainfully employed, the vast majority in the huge industrial concerns that have sprung up in that area, but many as maids and washerwomen in the houses of Europeans. Most of them are in a position to buy protein foodstuffs; though they are not by any means what one would call "well off", their diet approximates surprisingly closely to that of the better class of worker in Britain. Unfortunately, a

SECTION II - 3 (continued)

sizable chunk of their incomes is used in travelling the long way to work (an unfortunate affect of the Apartheid system), and almost as much is "invested" in the hire-purchase furniture stores - a fact incidentally, which in some parts of this town, is the prime cause of malnutrition amongst the children.

The second survey was undertaken in 1952 (Brinton, R.A. and Drysdale, B.E. (1952) J. Soc. Res. Vol 3, 114). These workers made a very careful study of four peasant groups widely spread round the country. One of their studies was round the town of Nongoma in Northern Zululand and embraces 47 families of what are referred to in this Thesis as "Rural Dwellers" or "country peasants". Nothing could be farther from the delusion often heard in this country, that the rural Zulu is"a fine specimen, well-fed, a great meat eater, and most sober and trustworthy". This unfortunately dates back to the days of Chaka, when the Zulus were indeed magnificent people. The truth now, unfortunately, is that they are under-fed, they only get meat if a beast dies of disease or is struck by lightning, or if they are able to poach one of the few unslaughtered wild antelopes in Zululand. Many Zulus have been contaminated by the habits of the Europeans,and alcoholism and venereal disease are both rife. The whole peasantry is firmly addicted to Dagga ("Insangu") - Cannabis indica, the illegal sale of which was, a few years ago before active police action, one of the few R10,000,000 agricultural businesses in the country. Almost all of the protein of the rural dwellers comes from vegetables, maize and pumpkin being the most common vegetables eaten. It is, in a way, fortunate that cattle are regarded as riches, and, consequently, milk is available in fairly reasonable quantities, though the unfortunate children only get what is left after firstly the father, and secondly his wives have drunk deep. To this very day, one cannot marry without paying the prospective father a certain number of cattle, proportionate to the obesity and social status of the bride. Almost all rural dwellers are brewers of alcoholic beverages, and drink large amounts, particularly of kaffir beer. This

SECTION II - 3 (continued)

is so rich in iron that if one were to drink a gallon daily (by no means an excessive intake), one can swallow iron equivalent to the total body stores in exactly one month. This is regarded as one of the most potent aetiological factors in the genesis of cytosiderosis of the liver. Incidentally, it is interesting to note that very few of the present series of diabetics were drinkers of kaffir beer.

The two diets are set out side by side in the following table :-

COMPARISON BETWEEN THE AVERAGE DAILY DIETARY INTAKES OF THE SETTLED URBAN ZULU AND OF 47 FAMILIES IN THE NONGOMA DISTRICT.

(Campbell, G.D. (1963). S.A. Med. J., 37,48,1199)

| ARTICLE OF FOODSTUFF | THE SETTLED URBAN ZULU | | CALORIFIC EQUIVALENT OF THE AVERAGE DAILY INTAKE OF VARIOUS FOODSTUFFS IN THE NONGOMA PEASANT. | |
|---|---|---|---|---|
| | CALORIES | % OF DIET | CALORIES | % OF DIET |
| CEREALS, (Maize and Maize products, and bread). | 1476 | 55 | 1376 | 47 |
| SUGAR | 419 | 16 | 30 | 1 |
| MILK AND MILK PRODUCTS | 112 | 4 | 224 | 8 |
| ANIMAL TISSUES<br>FATS AND OILS | 191<br>224 | 7) 15<br>8) | 40 | 2 |
| ALL VEGETABLES AND FRUIT | 268 | 10 | 1200 | 42 |
| TOTALS ..... | 2690 | ..... | 2870 | |

SECTION II - 3 (continued)

The chief differences between the diets of the town dweller and the country peasant are as follows :-

(1) As regards carbohydrate intake, the caloric value of which is remarkably similar in both groups, that of the peasant is almost 100% maize or maize product, home prepared. The town dweller eats large amounts of bread, particularly white bread, and Gampel, B., (1959)Pers. comm.)estimate that half the cereal intake of the town dweller is bread, and the other half maize or maize products. The vast majority of the present series were massive white bread-eaters before becoming diabetic.

(2) Sugar is hardly eaten at all by the country peasant. In certain parts of Northern Zululand, visited by myself in two Biological Expeditions in 1947 and 1949 (Maputaland), it was found every grain of sugar that did get into the area, is used exclusively in the concoction of various alcoholic beverages. Gampel, B. (1959 personal communication), reported the extraordinary fact that in their study of the Lamontville Location, they found that over 25% of all Zulu women over 45 living there, took no less than 105 or more teaspoons. This is PURE SUGAR INTAKE, as opposed to the large additional intake of sugar in jams, lemonades, and syrups of which they are so fond.

(3) The peasant eats large amounts of vegetables - in fact no less than 85% of his protein intake is of Plant origin.

(4) Contrary to popular belief, the Zulu peasant hardly ever gets meat at all. Most of his animal protein comes from milk.

(5) The peasant gets more in the way of milk and milk products than the urban dweller, particularly of "Amasa" - a most pleasant type of sour milk.

I performed a short dietary study upon one of my Zulu domestic servants, a lady aged 38 years who was grossly obese. She was permitted to order what food she wanted from the grocer, and I assessed what she chose. Her total caloric intake was 4580 cals. per day, and of this no less than 3900 (85%) of her calories came from white bread, sugar, jams and sweetened foods. (Campbell, C.D., Honeyman Gillespie Lecture, Univ. Edin., May 1966). This gives some idea of what is constituted in the mind of the urbanised Zulu

SECTION II - 3 (continued)

as "good food".

Per capita annual intakes of sugar expressed as an average per capita in pounds is of interest("Condenser". Publ. of the Tongaat Sugar Company, Maidstone, Natal.(1967). Vol. 5, 5, 15) and (S.A, Sugar Association (1968). Personal communication):-

| DATES | 1936 | 1943 | 1952 | 1959 | 1963 | 1967. |
|---|---|---|---|---|---|---|
| Whole Country | 40 | 70 | 76 | 79 | 79 | 80 |
| Rural Zulus | - | - | 4(i) 20(ii) | 39 | 60 | 55 |
| Urban Zulus | - | - | - | 87 | 86 | 75 |

(-) - Figures not available.

(i) - Abstracted from Brinton, R.A. and Drysdale, (1952). J. Soc. Res. 3, 114.

(ii) - Earlier S.A. Sugar Association figures (personal communication 1968)

These figures refer to (a) "direct" sugar intake, or sugar used as such added to food and drinks, and (b) "indirect" intake, which includes sugar incorporated in manufactured foods such as tinned fruit, jams and sweetened drinks.

The most interesting fall in consumption in both rural and urban Zulus has been due to price increases, and the method of the Zulu of purchasing sugar by monetary standards as opposed to weight. Thus they prefer to buy 10 cents worth of sugar, rather than 1 lb., and thus with price increases, the 10 cents-worth automatically gets less, resulting in diminished intake.

Intake in the rural dweller is difficult to interpret, as in some areas, such as Maputaland in Northern Natal, sugar is used solely in the manufacture of strong drink, chiefly in the "freshening" of the local palm wine. Rises in rural consumption have been striking, and are a measure of effective advertising

SECTION II - 3 (continued)

of the sugar authorities and the sweet manufacturers, and the purveyors of sweetened foods, chiefly jams and tinned fruit. The urban dweller may appear to have reached "nauseation point". This is a term first used by me (Campbell, G.D. (1963) E.Afr.Med.J.,5,272) to describe the theoretical ceiling of 110 lbs per head per annum (FAO) which is believed to be the highest intake that can be reached. This is no longer true, as the intake in Britain is 125 lbs per head per annum (International Sugar Council Year Book, 1967, 28, The Haymarket London. p. 32 ) and there are many races that eat far more than this figure. Indeed, I and a co-worker have recorded (Campbell, G.D. and Goldberg, M. (1966) S.A. Med. J., 40, 27 ) under the title of "The Sugar Orgy" a study of sugar intakes in cane cutters, where levels of 300 lbs per head per annum are exceeded by a group of people, chiefly Pondos (like the Zulus - Nguni people). It is tempting to try and link per capita sugar annual intake in any racial group to the possibility and extent of diabetes emergence in previously underprivileged groups. Thus in 1963 (Campbell, G.D. (1963), E.Afr. Med. J., 5, 272 ) I showed that one can relate what Prof. Tulloch refers to as "common" or "uncommon" incidence of diabetes to a borderline per capita annual intake of sugar of 70 lbs., by referring tropical countries mentioned by him (Tulloch, J. (1962). Diabetes Mellitus in the Tropics, E & S. Livingstone, Edin. and London, p. 21 ) to the figures of per capita annual intake as shown in the International Sugar Council Year Book (1963), (28 The Haymarket, London, p. 259). In this series comparing two groups - rural and urban Zulus, the per capita intake in the urban Zulus is above 70 lbs per head per annum, whereas in the rural dwellers it is still below. I, and my co-workers, have reviewed the relationship between sugar intake and diabetes fully (Campbell, G.D., Batchelor, E.L., and Goldberg, M.D. (1967) Diabetes, 16, 1, 62): we also discussed there people with very large intakes of sugar who are not diabetics, or perhaps more correctly, not yet diabetics.

## SECTION II - 4 RELATIONSHIP BETWEEN SOCIAL STATUS AND DIET INTAKE IN THE PRESENT SERIES.

There is no simpler method of assessing social status and income than by finding out how much maize or maize products Zulus eat daily, and how often they get protein supplements. Maize is the commonest crop grown by the peasants, but owing to its low cost and palatability it is also popular in the towns, and is eaten principally in a form of dry porridge called "putu", made of highly refined maize. Patients can, therefore, be divided into whether maize is a staple diet, and how often weekly the patient eats protein "supplements" (i.e. meat, fish, or eggs). There are three grades of diet and these are defined as follows :-

GRADE ONE: Maize is the staple diet, and patients have protein supplements less than once weekly. This is an extremely poor diet, and is responsible for the cases of pellagra seen in non-alcoholics. This is the diet that is eaten by large numbers of peasants and approximates to the peasant diet set out on page

GRADE TWO: In this diet maize is still the staple, but patients have protein supplements more than once weekly, but less than once daily. This is a fairly common diet in the town, especially amongst poorer urban labourers.

GRADE THREE: Maize is not the staple diet any more. Patients get protein supplements daily. This diet comes very close to the local European diet, except perhaps there is a higher carbohydrate intake. It is much the same as the diet of the better class of British worker.

In the following table is set by sexes, the number of diabetics in the present series that belong to the various dietary groups. Reference is made to this system on pages 42 - 43, where the dietary grades of the present series is compared with those of a number of siderotic patients, and the diets of the parents of the present series.

SECTION II - 4 (continued) TABLE NUMBER SIX.

NUMBER OF PATIENTS AMONG 133 ZULU DIABETICS IN THE VARIOUS DIETARY GRADES (after Lamont) SHOWING TYPE OF DIABETICS AND TYPICAL OCCUPATIONS OF THE VARIOUS DIETARY GRADES.

| DIET | OCCUPATION | S = SENILE M/A = MIDDLE-AGED Y = YOUNG | | |
|---|---|---|---|---|
| | | MALE | FEMALE | TOTAL |
| GRADE ONE: Very poor diet MAIZE is the staple diet. Patients have supplements (meat, fish, eggs) LESS THAN ONCE WEEKLY. The classical "pellagra" diet in non-alcoholics. | PEASANTS URBAN INDIGENTS | S - | - | - |
| | | M/A - | - | - |
| | | Y - | - | - |
| GRADE TWO: MAIZE is still staple. Patients have supplements MORE THAN ONCE WEEKLY | WASHERWOMEN, PEASANTS, URBAN LABOURERS, INHABITANTS OF THE URBAN "SHANTY TOWNS". | S 3 | 8 | 11 |
| | | M/A 9 | 32 | 41 |
| | | Y 5 | 4 | 9 |
| GRADE THREE: MAIZE is NOT staple any more. Supplements are eaten daily. Diet very like that of the European with slightly higher CHO intake. | BUSINESSMEN TEACHERS, CLERGYMEN, INDUNAS, STALLHOLDERS, S.A. POLICEMEN HOSPITAL WORKERS POSTMEN, TAXIMEN, WHITECOLLAR WORKERS, WIVES OF RICH MEN, SCHOOLMASTERS. HOUSING SCHEME DWELLERS | S 2 | - | 2 |
| | | M/A 23 | 28 | 51 |
| | | Y 11 | 8 | 19 |
| | TOTALS | 53 | 80 | 133 |

SECTION II - 4 (continued

REFERENCES TO METHODS OF DIETARY GRADING -
Gillman, T., Hathorn, M.K.S. and Lamont, N.McE. (1961). Extrait De acta Union Internationale Contre le Cancer (Belgium). Vol XVll, Nos. 5 - 6, Chapter 5, 1.
Gillman, T., Hathorn, M.K.S., and Lamont N.McE. (1958) S.Afr. J. Med. Sci., 23 , 187.

This table shows that in the present series not one diabetic came from the Grade ONE (or "pre-pellagra") diet, that is, the diet that appears to contribute so much to the syndrome of cytosiderosis in the Bantu. No less than 54% of these patients lived on a Grade Three diet - approximating to that of the Natal European. The rest belonged to the Grade Two Diet category, one that is particularly prevalent amongst the middle class Zulu, composed of washerwomen and urban labourers. The table shows the various occupations of the dietary groups, and that low maize intake is a good index of higher worldly status.

## SECTION II - 5 ALCOHOL INTAKE IN DIABETIC AND NON-DIABETIC ZULUS.

The Durban workers, Lamont, Gillman and Hathorn have since 1959 forcefully advanced the thesis that the syndrome of cytosiderosis in the Bantu is intimately related to kaffir beer intake. In support of this theory, they reported the case of a Natal Indian, addicted to this beverage (most unusual to one who has legal access to European drink), who developed siderosis - a most unusual syndrome in the Indian. (Lamont, N.McE., Gillman, T. and Hathorn, M.K.S. (1959) Brit. Med. J. i, 1391).

In the present series of diabetics, at my request these workers performed liver biopsies on a small series of 16 diabetics, and in this series, siderosis was found in only two patients. Consequently, it is most relevant to review the kaffir beer intake of the diabetics in this series. This intake is compared to certain observations made by Gampel, B. (1959) (Personal communication) on the beer intake of non-diabetic Zulus living in the Lamontville Location.

SECTION II - 5 (continued)

TABLE SHOWING THE PERCENTAGE OF ZULUS WHO DO DRINK KAFFIR BEER.

| | NON-DIABETIC URBAN SETTLED ZULU. | ZULU DIABETICS. |
|---|---|---|
| ALL FEMALES OVER 18 | 20% | 2% of 80 patients |
| ALL FEMALES OVER 45 | 31% | |
| ALL MALES | 68% | 42% of 53 patients. |

The Zulu is an honest patient and will discuss alcoholic intake freely and frankly: The above figures are perfectly valid, which is perhaps more than one could say for an equivalent series of European patients! The table shows that the diabetics, especially the females, do not drink nearly as commonly as their non-diabetic counterparts. Whether the low incidence of cytosiderosis is due to this fact or to the better diet, is very hard to say.

In the present series of diabetics, no male or female patients admitted to drinking the illicit liquor "gavine", which is a distillate made from certain fermentates to which often toxic substances such as carbide might be added for "effect". It is not easy to judge the effects of the general release of "European" liquor to the Zulu people, which took place about 1964, in the people in the present series, excepting to say that when these "European" drinks (gin, vodka, brandy, brandy-wine mixtures and ordinary beer) were released, we redoubled our efforts in the Clinic in all patients, whether Indian or Zulu, to try and advise strongly against drinking generally. In a recent bottle store survey in Zululand (Campbell, G.D. (1967) "The African as Insurance Proponent in Southern Africa", unpublished address to the 1967 Congress of the S.A. Medical Association, Durban) I became alarmed at the amount of these liquors that were being consumed by the working class people on the sugar belt, and especially amongst people who could ill afford their expense.

## SECTION II - 6 COMPARISON OF THE DIETARY GRADINGS OF THE PRESENT SERIES AND THOSE OF THEIR PARENTS - "THE GREAT LEAP FORWARD" OVER ONE GENERATION.

Dietary gradings after the method of p. 38, of the following groups are set out in the following table: (i). The present series of Zulu diabetics. (ii). The parents of 104 of the present series of diabetics. (iii). A series of 200 consecutive ward patients on whom liver biopsy was done(believed to be cytosiderosis cases). (iv). A control group of 237 consecutive ward admissions(male Zulus, medical wards), and (v) a series of 100 patients suffering from porphyria.

(ALL EXPRESSED IN PERCENTAGES)

| | Grade 1 (poor) | Grade 2 | Grade 3 (good) |
|---|---|---|---|
| Present series of 133 Zulu diabetics. Males | 0 | 13 | 24 |
| Females | 0 | 33 | 30 |
| | 0 | 46 | 54 |
| Dietary gradings of parents of 104 of the present series of diabetics. | 51 | 26 | 23 |
| Other Groups:- 200 cases suspected of being siderotic. Liver biopsies:- 88% siderotic: 75% severely siderotic: (Gillman, T., Hathorn, M.K.S. and Lamont, N.McE. (1961) Extrait De Acta Union Internationale Contre le Cancer (Belgium) Vol. XVII, Nos. 5 - 6, Chapter 5, 1.) | 68 | 28 | 4 |
| A control series of 237 consecutive male Zulu admissions to the Ward. (Lamont, N.McE. (1959) Pers.comm.) | 54 | 37 | 9 |
| A series of 100 patients proven as suffering from porphyria. (Lamont, N.McE., Hathorn, M.K.S. and Joubert, S.M. (1961). Q. J. Med. (New Series) Vol.30, 120, 370. | 35 | 61 | 4 |

The differences between the diabetics and their parents is significant ($p < .01$) and the difference between the diabetics and the siderotics highly significant ($p < .001$).

SECTION II - 6 (continued).

There are some remarkable differences in the dietary gradings of the various groups of people which can be summarised thus :-

(1) The diabetics were overwhelmingly well fed, and in such people protein intake is a good guide of money available for all foods, this is not surprising.

(2) Nutrition in the diabetic series was very much better than in the case of their parents.

(3) The siderotics had a very poor intake of protein indeed, and their intake was similar to that of the general patients in the wards of a large charity hospital.

(4) Porphyria in Durban is closely linked with the ingestion of illicit distilled liquors, which are quite expensive. Thus it is most interesting to note that food intake in this group was somewhat better than the siderotics, but a good deal inferior to that of the diabetic series. This shows that they had enough in the way of wages to indulge in the purchase of these illicit brews (chiefly "gavine") to contract porphyria. (Lamont, N.McE., Hathorn, M.K.S. and Joubert, S.M. (1961). Wld. J. Med. (New Series) Vol. 30, 120, 376).

SECTION II - 7 FAMILY HISTORY OF DIABETES.

It has been most uncommon to find in the present series of 133 diabetics, or indeed in any 133 diabetics, a family history of the disease, with the notable exception of the Zulu Royal Family who are detailed below. The following table shows the number of patients who have actually had a family history of diabetes at the time they were first seen.

| | EITHER PARENT | SISTER OR BROTHER | CHILDREN | AUNT or UNCLE | TOTAL |
|---|---|---|---|---|---|
| NUMBER OF PATIENTS WITH FAMILY HISTORY AT FIRST VISIT. | 2 | 2 | - | - | 4/133 |

SECTION II - 7 (continued)

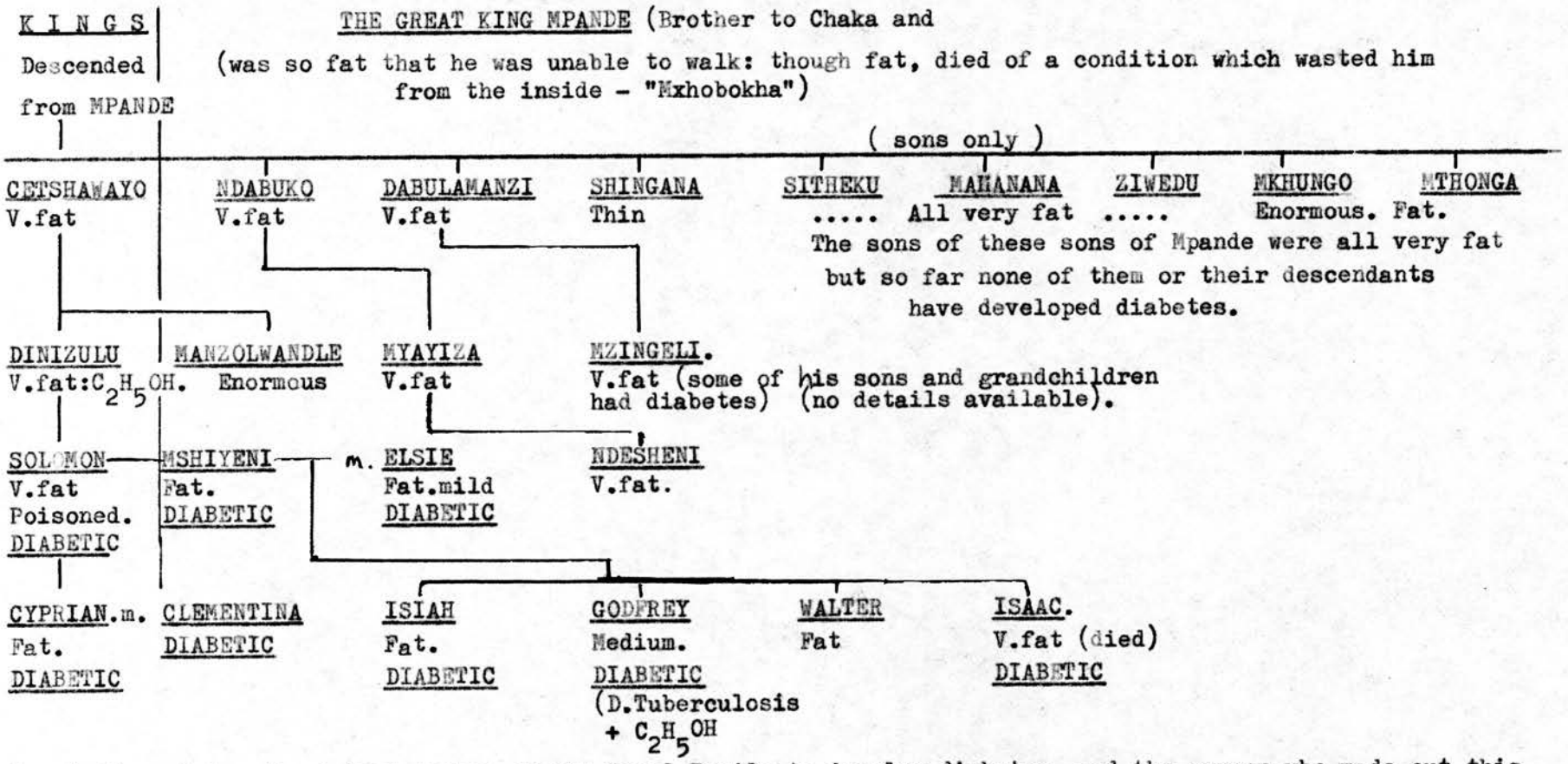
THE ROYAL FAMILY (The House of Zulu)

It is extraordinary how many Royal families in history have been afflicted with certain diseases. As diabetes is so rare in the rural Zulu, it is noteworthy that so many of the Zulu Royal Family suffer from the disease. Indeed they are the only family in which I have been able to find a really comprehensive family history amongst the Zulu people. Recently the disease has also been shown to be common in the Basuto Royal Family. As stated above, I believe that the diabetes in the family starts from the massive King Mpande who was so obese, he was unable to walk.

Through the passage of the last 100 years few Zulu families have had it as good economically as the Royal Family. Invariably, whether in office or in exile in St. Helena (Dinizulu - "The Black Napoleon"), they have been over-nourished, and though enormously fat Zulus are not frequently seen in the streets of Durban, "it is true to say that intimate members of the Royal Family, until recently, were readily recognised by their enormous obesity" (Zulu, G. 1960, Personal Communication).

The following family tree shows how significant numbers of the descendants of Mpande, more particularly the elder sons, have become diabetic, and principally those of his exiled grandson, Dinizulu, mentioned above. It is of interest to note that both the late Regent Mshiyeni ka Dinizulu, and the present King, Cyprian ka Zulu, have both married diabetic women.

## GENEALOGY OF THE ZULU ROYAL FAMILY, WITH REFERENCE TO OBESITY AND DIABETES




Mr. Godfrey Zulu, the latest member of the Royal Family to develop diabetes, and the person who made out this family tree, said in his letter - "It is true to say, that intimate members of the Royal Family until recently, were readily recognisable by their enormous obesity". (Zulu, G. 1959 - Personal Communication)

SECTION II - 7 (Continued)

Follow-up Cases:

Case No. 97. A 52 year old male policeman was of interest, in that during his five years of attendance at the Clinic he reported a family history of diabetes in that one brother and one sister, both younger than himself, developed the disease.

CONNUBIAL DIABETES.

My Clinic is well-known because of the enormous number of what I have called "Connubial diabetics", i.e. husband and wife diabetic pairs. (Campbell, G.D. (1961) Brit. Med. J., 2, 1538). Of the 309 such pairs registered, seven were Zulu connubial diabetic pairs, but there was only one woman in this series (Case No. 96) whose husband had been diabetic, and as the children were scattered around the country it was not possible to study them. In passing, I and my co-workers have noted that it is likely that having connubial diabetic parents does not increase the possibility of children being diabetic. (Goldberg, M., Campbell, G.D. and Jackson, W.P.U. (1967) Exerpta Medica No. 140, 53).

FAMILY HISTORY OF OTHER ENDOCRINE DISEASES.

Family history of other endocrine disease is absent in the present series. Endocrine disease on the whole is very uncommon in the Zulu people. Thyroid disease is confined solely to the presence of non-toxic nodular goitres on the basis of iodine-deficiency goitre and the parenchymatous goitre of puberty. Thyrotoxocosis is almost unknown in the Zulu. At present in the Diabetic Clinic there are about 250 goitre patients, half being Zulus, none of whom is toxic and all of them have the iodine deficiency goitre which has gone on to nodulation at an early age. None of our goitrous patients are diabetic. One of the present series of diabetic patients has had thyroidectomy for a large non-toxic nodular goitre, and the indication for thyroidectomy was because of local pressure from the enlarged gland. Another patient (p. 102) disappeared when we asked her to submit to operation for a large isthmic goitre.

SECTION II - 8

## THE "RULE OF 20 YEARS".

It was noted, in the course of taking histories from the present series of patients, that many of them had lived in the town for 20 years, or a period of time close to this, before developing the disease. (Campbell, G.D. (1959), Congr. S.A. Med. Assocn., East London. Brochure of Papers, 45).

An attempt was made to try and establish exactly how much this aetiological "Rule of 20 Years" applied to 80 fat middle-aged diabetics, whose duration of disease was between 0 - 12 years, and had come from the country to live in the town. The following table shows the "periods of exposure" by two year periods in these patients before the diabetes became manifest.

(see table on following page)

TABLE SHOWING THE LENGTH IN YEARS OF "EXPOSURE TO BIG CITY LIFE" IN 80 FAT DIABETICS BETWEEN THE AGES OF 36 AND 62 YEARS, TAKEN IN (TWO YEAR) PERIODS OF "EXPOSURE" FROM 4 - 36 YEARS.

| | 5. 6. | 7. 8. | 9. 10. | 11. 12. | 13. 14. | 15. 16. | 17. 18. | 19. 20. | 21. 22. | 23. 24. | 25. 26. | 27. 28. | 29. 30. | 31. 32. | 33. 34. | 35. 36. | 37. 38. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| MALES | - | 1 | - | 1 | 2 | 2 | 3 | 5 | 5 | 1 | 1 | - | 3 | - | 3 | 3 | |
| FEMALES | 3 | 2 | 1 | - | - | 2 | 2 | 13 | 9 | 5 | 2 | 2 | 2 | - | 2 | 5 | |
| TOTALS | 3 | 3 | 1 | 1 | 2 | 4 | 5 | 18 | 14 | 6 | 3 | 2 | 5 | - | 5 | 8 | |

This table is interesting in that no less than 53% of all our fat diabetic patients (ages 36 - 62) who came to live permanently in Durban, developed the disease between after 17 and 24 years "exposure". In order to see whether this tendency was spread over the whole age range (36 - 62 years), the two curves were made, one for those under the age of 50, and one for those over the 50 age, and these showed a similar form.

SECTION II - 8 (continued)

As many of the present series were new diabetics in 1958 - 1959 when the collection of patients was made, the application of the "Rule of 20 Years" which supposed that many of them came to the City, the years just before the Second World War, when it was popularly believed there was a large influx of Zulus into Durban. This is not true according to registration figures in the Durban Native Administration Department, (1960 Personal Communication), which shows that the biggest increases in registrations per year came in the immediate post-war years 1946 - 1948. Why, therefore, the "Rule of 20 Years" should apply particularly to immigrants in the immediate pre-war period is hard to say.

However, it is particularly interesting to note how the majority of patients registered in the present series (having been collected in 1959 and having been in town for 20 years) had moved into Durban between 1936 and 1943, as this coincided with a large and dramatic increase in national sugar consumption. In 1936 ("Condenser" (1967). Publ. of the Tongaat Sugar Company, Maidstone, Natal, Vol. V, 5, 15) the national per capita intake of sugar was averaged at 40 lbs., whereas in 1943 it had risen to over 70 lbs, that is to say, it had almost doubled in the space of 7 years. This was due to the recovery from the depression, to the spectacular sales of brown sugar, and to the near-boom conditions that obtained in Durban during the first part of the War, when huge numbers of ships used the Port on the way to the East, and this increased the purchasing power of the urban Zulus enormously. Recent trends in sugar consumption locally are set out on page 36.

Since this observation was made and drawn to the attention of other workers, it is interesting to note that both Cohen in Israel (Cohen, A.M. (1960) Israel Med. J. 19, 6137) and Albertsson in Iceland (Albertsson, V. (1953) Diabetes 2, 184), have noted periods of fairly constant exposure to diet in immigrants before developing diabetes, and Cohen has actually extended this observation to the emergence of myocardial infarction.

SECTION II - 9

ABILITY TO SPEAK ENGLISH AS FURTHER EVIDENCE FOR SOCIAL EMERGENCE.

It is always held that ability to speak English is generally a sign of social advancement in the Zulu people.

| | ENGLISH SPEAKING | SEMI-ENGLISH SPEAKING | ZULU SPEAKING ONLY |
|---|---|---|---|
| PRESENT SERIES OF 133 ZULU DIABETICS | 41 | 42 | 50 |
| CONTROL GROUP OF 133 ZULU OUT-PATIENTS. (over age 25 :- 83 females, and 50 males.) | 29 | 40 | 64 |

("English speaking" refers to those fluent in English, and "Semi-English Speaking" to those speaking with difficulty.)
This table shows that there is only the very slightest difference between diabetics and non-diabetics as regards their ability to speak English, and these differences are not significant. (p. not $<.05$)

SECTION II - 10 LITERACY RATE (ABILITY TO READ ENGLISH AND/OR ZULU.

| | FULLY LITERATE | SEMI-LITERATE | ILLITERATE. |
|---|---|---|---|
| PRESENT SERIES OF 133 ZULU DIABETICS | 39 | 46 | 48 |
| CONTROL GROUP 133 ZULU OUT-PATIENTS (over age 25: 83 females 50 males) | 19 | 29 | 85 |

Most of our patients were illiterate or semi-literate, but the illiteracy rate of the same control group as in Section II - 9 was striking.

PART III - THE CLINICAL DIABETIC STATE IN THE PRESENT SERIES.

SECTION III - 1 GROUPS OF DIABETICS IN THE SERIES.

The basic diabetic nomenclature used in my Clinic have been set out in an introductory section on p. 21, with the number of diabetics in each group. In Scetions III - 2 to III - 6, are details relevant to the present discussion about the following diabetic groups:-

Section III - 2. The Senile Diabetic.
3. The Middle-aged Diabetic.
4. The Young Insulin-dependent Diabetic.
5. The so-called "J" type of Diabetic.
6. The Insulin-independent Young Diabetic.

SECTION III - 2 THE SENILE DIABETIC.

Those diabetologists who still use the Terms "Type1", "Type 11", "Lipoatrophic", and "Lipodystrophic", apart from introducing elements of confusion by using code and non-descriptive names, lose sight of the fact that there is an important groups of diabetics who develop the disease in old age. Onset is generally at an age of over 60 years: many are thin, and however well any may restrict carbohydrate intake, drug treatment, whether by one of the oral agents or by means of insulin, is almost invariably necessary. Some patients, even need large doses of insulin(Case 116), and certainly some need much larger doses than might have been expected.

In the present series there were 13 out of a total of 133 who were "senile" diabetics, and all were country peasants, excepting one (Case 133) who was a Parson living under good social conditions. The predominance of country-dwellers in this group was at one time so marked, that I suggested the name "Peasant" diabetics for them(Campbell, G.D. (1959). Ann. Rep. M.O.H. Durban, Appendix III, 2). In this context it is interesting to note that other workers(Shaper,A.G. (1957). Proc. 3rd Congr. Intern Diab. Fedn., Editions Medicine et Hygeine, Geneva. 781) have also commneted upon the preponderance of peasants in other series of diabetics in Africa. The Clinic reference numbers of Senile Diabetics in the present series are set out on p.152. There is little doubt that clinical considerations make the group of "senile" diabetics valid in the Indian and European populations as well as in the Zulu(Campbell, G.D. (1961). Med. Dig.(Bombay) 29, 714) As most of these patients are illiterate it is difficult to instil into them an insight into the potential seriousness of their condition,

and the lamentable degree of follow-up amongst these admittedly few patients emphasises this(p. 134 ).

It may be possible by the insulin-dependent state of some of these patients that their diabetes may be due to exhaustion of their islet cells, possibly as a result of interference of their vascular supply, and this would put them into the category of truly insulin-dependent diabetics. However, many are satisfactorily controlled upon insulin and hypoglycaemic agents, and some on the sulphonylureas, which are now generally regarded as being able only to work in the presence of intact islet cells. Such conjectures would be difficult to prove in the absence of careful necropsy findings taken into account with plasma insulin levels before and after glucose loads,and the clinical fainings. Their thinness is the most important clinical factor that distinguishes them from the middle-aged group of diabetics.

THINNESS AND OBESITY IN 13 SENILE DIABETICS.

| | THIN | FAT | TOTALS. |
|---|---|---|---|
| MALES(5) | 4 | 1 | 5 |
| FEMALES(8) | 6 | 2 | 8 |
| TOTALS | 10 | 3 | 13 |

Beacuse of their illiteracy, one is very much more solicitous of treatment in the senile group than in the others. In one way, control may be considered satisfactory in that they are not repeatedly admitted to Hospital in ketosis, though the withholding of insulin from those apparently needing it results in heavy glycosuria, loss of weight and severe ill-health.

SECTION III - 3 THE MIDDLE-AGED DIABETIC.

More than half of the patients in this series were in the group of middle-aged diabetics - 88 out of the 133: and of the 88, 56 were females and 32 males. Clinic reference numbers are set out on p.149 .

This group would approximate to what are referred to as the "maturity-onset" diabetics. Ages of onset are mostly between 40 and 60 years of age, but there are some patients in their thirties whose clinical state(extreme obesity and independence from insulin) which put them into this category.

Of the 88 patients in this group, 80 were considered "fat" and 8 were considered"thin". As there exist no tables for "normal" weights in the

SECTION III - 3(continued)

Zulu people, the division bteween fat and thin was made on clinical grounds, with a special attempt to say that patients were either fat or thin. Breakdown by sexes was as follows:-

THINNESS AND OBESITY IN 88 MIDDLE-AGED DIABETICS.

| | THIN | FAT | TOTALS |
|---|---|---|---|
| MALES | 2 | 30 | 32 |
| FEMALES | 6 | 50 (see p. 48 - The Rule of 20 Years) | 56 |
| TOTALS | 8 | 80 | 88 |

The Zulu people and especially the women gain large amounts of weight when they migrate into the towns, and the degree of obesity seen in these females can be seen to exceed that of women in the New World. (Slome, C., Gampel, B., Abrahamson, J.H., and Scotch, N.(1960). S. Afr. med. J., 34, 505). Earlier, I set out(p. 35) the possible role played by the ingestion of refined carbohydrate in the genesis of diabetes and obesity. In the light of this, it is very interesting to study the eating habits or rural-dwellers not suffering from diabetes, when they are admitted to the General Medical Wards of the Hospital(Lamont, N.McE. (1960). Pers. comm.). These people eat as much as they possibly can when they are admitted to the Wards. This hyperphagic phase lasts for about 7 - 10 days, after which they seem content with a single large helping of food for each meal.

Connotations of treatment in the middle-aged diabetic in this series are the same as in more temperate climates: when patients can restrict carbohydrates, they respond well, with relief of symptoms and lessening of glycosuria. They seldom show ketones in their urines, and with very few exceptions, do not depend upon insulin for good health(p.55 ). They respond well to the oral agents, but the sulphonylureas make them gain weight(Campbell, G.D., McKechnie, J., Brokensha, B., and Davidson, J. (1960). Trial of a New Anorectic Agent - "Cleofil". Unpublished work.) With the biguanides, patients respond well, and at the same time they lose weight. In the middle-aged patients, symptoms are worst at nights - particularly nocturia, which results from large evening meals eaten

SECTION III - 3(continued)

by tired patients who have often travelled a long way home from their work. Thus it is very important that restriction of carbohydrate at their evening meals can be stressed, as by doing so, this distressing symptom may be largely avoided.

The middle-aged often fat diabetic is now swamping the young insulin dependent patients in the Clinic, and in comparison the number of senile diabetics is very low: it would appear certain that in the years to come, that we will see large numbers of these diabetics, in view of the rapidly-rising purchasing power of the Bantu(African) people, especially in the urban areas of this country.

SECTION III - 4 THE YOUNG INSULIN-DEPENDENT DIABETIC.

There were 32 young insulin-dependent diabetics in the series, 16 being males and 16 females(Clinic reference numbers, p. 148). These patients are similar to the insulin-dependent diabetics seen in Europe or America, in that they are thin, suffer from a labile diabetic state, and lapse readily into ketosis if insulin is withheld. One difference is the comparative flexibility of insulin dosage, as in these people, insulin dosage can be approximated to the nearest 10 units of insulin, and there does not seem to be the same necessity for more precise control - something that is probably engendered by our chronic fear of severe hypoglycaemic attacks(p. 96). That at the end of 9 years of follow-up, no less than 10 of these 32 patients are still attending is perhaps one of the most remarkable records of this thesis. Of the female patients, 4 were "not thin"(Cases 27, 118, 120 and 142), but they became rapidly ketotic when insulin was stopped. There were 2 cases(Nos. 4 and 14) who were the original cases of the "insulin-independent young diabetics of the Tropics", described by me (Campbell, G.D.(1960) Brit. med. J., ii, 537), in whom control of diabetes in pregnancy was so bad, and the patients forgot to take their insulin for long periods, that they were changed, in desperation to oral treatment, with satisfactory control! They are discussed in more detail on p. 56. Thus it would appear, and we have since confirmed this, that young girls with established diabetes in Tropical or sub-tropical areas, who can go through pregnancy without needing insulin, even when they are thin.(see p.129).

In contrast to these insulin-independent young diabetics, are certain fat patients, often over 40 years old at onset, where the oral agents

SECTION III - 4(continued)

do not succeed, and where the patient appears to be truly dependent upon insulin for the maintenance of good health, even though ketones have never been seen in the urine:-

Case No. 7 was a 48-year old female at onset of diabetes, when she weighed 208 lbs, having, as she put it, "lost a lot of weight". She was treated with all the oral agents we had at our disposal(tolbutamide, metahexamide, chlorpropamide, acetohexamide, phenformin) alone or in various combinations, even in maximum dosage, and responded to none of these measures. In spite of never having had ketones in her urine she steadily lost weight, and became unwell. When her weight had fallen from 208 to 128 lbs., she was put on to Lente insulin, 40 units daily, and on this dose she regained her previous good health, but she continued to gain weight inexorably, so much so that within 8 months after starting her insulin she was back to 200 lbs.again. Further attempts to take her off insulin, which she resists vigourously, are invariably associated with sudden weight loss, which is rapidly restored by putting her back on insulin again. Needless to say, I tried to find out what complicating factors might have been responsible for this, but chest Xrays were invariably normal, and urine cultures always sterile. She now refuses further attempts to wean her off insulin, though she would dearly like to take "the pills". In this account, her name is included in the category of middle-aged diabetics, though, apart from her age, she is probably a person that can be included with justification in the category of insulin-dependent diabetics. The body state of the insulin-dependent diabetics was:-

THINNESS AND OBESITY IN THE YOUNG INSULIN-DEPENDENT DIABETICS

| | THIN | FAT | TOTALS |
|---|---|---|---|
| MALES | 16 | - | 16 |
| FEMALES | 12 | 4 | 16 |
| TOTALS | 28 | 4 | 32 |

In conclusion, this group of insulin-dependent patients contains some of my most conscientious patients, many of whom are intensely grateful

SECTION III - 4(continued)

for what we have been able to do for them.

SECTION III - 5 THE SO-CALLED "J" TYPE OF DIABETIC.

Though in 1959,(Campbell, G.D. and McNeill, W.G.(1959). Brit. med. J., ii, 634) we felt inclined to believe that such a "Tropical variant" of diabetes did occur in my Clinic, subsequently, we believed that it did not. The best way to regard claims for the existance of this variant - believed to be a feature of diabetes in warmer climates - is to view such claims in the light of the fact that no less than 3% of patients attending Diabetic Clinics in the United Kingdom can be said to fall into this "type"(Kellock, T.D.(1962). Abstr. Wld. Med., 31, 49). The variety was first described from Jamaica(Hugh-Jones, P.,(1955). Lancet ii, 981). Subsequently, simultaneous papers from Jamaica(Tulloch, J.A. and McIntosh, H.D.(1961). Lancet, ii, 119) and Natal(Campbell, G.D. (1961). Med. Proc., 7, 19, 395) showed that this variant was almost certainly not valid. Thus I do not intend to discuss it further here. Another variant, the "K" type of diabetic was described in 1957, (Shaper, A.G.(1957). Proc. 3rd. Intern. Congr. Intern Diab. Fedn., Editions Medicine et Hygeine, Geneva. 781). This is also not believed to be valid, as the patients that fall into this "K" as well as the "J" type all fall much more lucidly into the descriptive nomenclature that I set out on p. 24. I believe most strongly that it is far better to cling to descriptive rather than codal nomenclature.

SECTION III - 6 THE INSULIN-INDEPENDENT YOUNG DIABETIC.

In 1960, on the basis of the 2 patients described on p.129 who went through pregnancy on the oral agents and a number of other young female diabetics in the Clinic, I suggested the name "insulin-independent young diabetics" to describe these unusual patients. In these people, onset was generally under the age of 25 years and often under 20 years, onset was often acute, with marked diabetic symptoms and heavy glycosuria, many were fat, though there were about one-quarter who were thin, who never showed ketones in their urines, but who responded well, even in pregnancy to the oral agents.(Campbell, G.D.(1960). Brit. med. J., ii, 537). I had actually drawn attention to these patients in a less well-known publication in 1959(Campbell, G.D.(1959). Med. Proc., 5, 26, 599).

These patients spotlight one of the most important differences between diabetic practice in Europe and America, as compared with that in the

SECTION III - 6(continued)

Tropics and Sub-tropics, and that is the remarkable absence of ketosis in certain young diabetics of acute onset, and a good response to the oral agents. In this series, only 2 cases(Nos. 4 and 14), who were included in the original breakdown as patients needing insulin, came into this category, and they are discussed in detail on p. 129. To illustrate the remarkable cases seen on occasion in my Clinic, I would like to divert and describe briefly the case of a young Natal Indian(Mohamed Sidiq), who was the child of one of connubial diabetic pairs. He developed the classical symptoms and signs of diabetes at the age of 18 months which were easily recognised by his mother who confirmed the diagnosis by finding his urine loaded with sugar. He was actually referred by a country doctor, on treatment with 500 mgms.(!) of chlorpropamide daily, which he had been taking for a full month without any hypoglycaemic effects. Treatment was stopped, with return of symptoms and heavy glycosuria, and a 50-gm glucose tolerance test confirmed the presence of diabetes . On no occasion were ketones ever present in the urine. The intensive study of such cases would be most rewarding.

SECTION III - 7 THE AGES OF ONSET OF DIABETES, AS FAR AS COULD BE JUDGED FROM THE HISTORY.

THE AGE OF ONSET BY SEXES IN 133 ZULU DIABETIC PATIENTS, TAKEN IN FIVE-YEAR PERIODS FROM 10 - 85 YEARS.

| AGES OF ONSET | 10. 15. | 16. 20. | 21. 25. | 26. 30. | 31. 35. | 36. 40. | 41. 45. | 46. 50. | 51. 55. | 56. 60. | 61. 65. | 66. 70. | 71. 75. | 76. 80. | 81. 85. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| MALES (53) | - | 1 | - | 2 | 6 | 8 | 9 | 13 | 6 | - | 5 | 1 | 1 | 1 | - |
| FEMALES (80) | 2 | 2 | 2 | 6 | 7 | 8 | 12 | 12 | 10 | 11 | 3 | 1 | 2 | 1 | 1 |
| TOTALS (133) | 2 | 3 | 2 | 8 | 13 | 16 | 21 | 25 | 16 | 11 | 8 | 2 | 3 | 2 | 1 |

Only five patients were under the age of 20 when they developed diabetes. Though it will be seen that actually 16 patients had ages of onset of over 60 years, these were not all included in the category of senile diabetics, as three of them were grossly overweight urban dwellers who were just over 60 when first seen, and had more of the factures of the middle-aged type of patient, and were included in that group. Most of the patients know their ages, or approximate birth years. Those that did not were asked a series of questions (Campbell, G.D. and Lugg, H.C. (1961) Handbook to Aid in the Treatment of the Zulu Patients; page 11 on the "Age of the Patient") to try and relate their births or earliest memories with certain events that took place. The chief of these are :- (a) 1884 - Cetshawayo's death (b) 1895 First locust invasion (c) 1897 The Rinderpest of cattle (d) 1899 - Beginning of the Boer War (e) 1906 - Zulu rebellion of Bambata (f) 1910 Halley's Comet, and the Act of Union, and (g) 1914 - the Great War begins.
Patients born subsequently to these dates generally knew their ages accurately.

## SECTION III - 8 SYMPTOMS AT ONSET OF THE DISEASE.

The Zulu diabetic presents clinically with very much the same symptoms that herald the disease in the European patient. In the following table are shown the percentages of the present series who complained of the ten commoner symptoms at the onset of diabetes :-

TABLE

SHOWING IN 133 ZULU DIABETICS THE COMMONER SYMPTOMS COMPLAINED OF WHEN FIRST AFFLICTED WITH DIABETES

(Expressed in Percentages.)

| | | |
|---|---|---|
| DIABETIC COMA | 7% (first admission only: others came back in coma.) | (9 patients - 2 males, 7 females) |
| POLYURIA | 98% | |
| POLYDIPSIA | 98% | |
| WEIGHT LOSS | 92% | |
| DIMNESS OF VISION (and other eye complaint) | 76% | |
| IMPOTENCE | 66% of 50 males. | |
| PRURITUS | 54% - Females 50%<br>Males 5% | |
| PAINS IN THE LEGS | 50% | |
| AMENORRHOEA (and menstrual upset). | 34% of 48 menstruating females. | |
| DYSURIA | 34% | |
| SKIN INFECTIONS | 10% | |
| Non-specific complaints, such as weakness and tiredness, were not included in the table. | | |

Most of these symptoms are discussed in detail in the sections on Diabetic Complications. Notable features were (a) that asymptomatic diabetes was extremely rare in the series and (b) the high incidence of impotence seen. This is not surprising to doctors who worked amongst the Zulu people, the males reacting very quickly to any generalised disease, such as Tuberculosis, by becoming impotent.

## SECTION III - 9. THE CLINICAL EXAMINATION

Deatils of procedure of examination are set out in full on p.112 .

SECTION III - 10 DIABETIC COMPLICATIONS AND THREE MAINSUBDIVISIONS.

For the sake of convenience, the diabetic complications have been divided up into three groups. Firstly, those that pertain primarily to the Vascular System: secondly, those complications that are Infective Complications: and, thirdly, those that might be regarded as being predominantly "Metabolic" Complications. These groups and the complications pertaining to them are set out :-

(a) VASCULAR COMPLICATIONS.

1. Hypertension, Cardiac Enlargement and Congestive failure.
2. Cerebral Vascular Disease.
3. The Diabetic Nephropathy of the Kimmelstiel-Wilson type.
4. Vascular changes of the retina.
5. Peripheral Vascular Disease.

(b) INFECTIVE COMPLICATIONS.

1. Tuberculosis.
2. Infective Nephropathy, and all Urinary Tract Infarctions.
3. Pruritus Pudendi.
4. Abscesses of the skin, and breast abscesses.

(c) METABOLIC COMPLICATIONS.

1. Subjective diabetic Neuropathy.
2. Objective Neuropathy.
3. The Senile Diabetic Cataract.
4. The "true" or juvenile diabetic cataract.
5. Liver enlargement.
6. Diabetic coma.
7. Hypoglycaemic coma.

In subsequent sections I have detailed tables which attempt to relate the duration of diabetes with the onset of Vascular Infective or Metabolic Complications. "Duration" is how long the patient (or his doctor) has known that he had diabetes.

In these tables, in accordance with my intention of studying incidence of complications in these diabetics, the figures refer to what was found between the time the patients were first seen and at any time during the minimal follow-up of one year. Patients developing complications subsequently are noted in the appropriate sections under "Follow-up Cases". In instances where large numbers of patients

SECTION III - 10 (continued)

suffered from various complications (Subjective Neuropathy 51.0% or Pruritus Pudendi 36.8%) case reference numbers have not been set out.

COMPOSITE TABLE OF ALL COMPLICATIONS AND CONCOMITANTS IN 133 ZULU DIABETICS (Duration of Diabetes 0 - 12 years) WHEN THEY WERE FIRST SEEN OR UP TILL THE END OF THE FIRST YEAR'S FOLLOW-UP.

| | Onset of Diabetes (New cases) | 0 - 5 yrs. | 5 - 10 yrs. | 10 yrs. Plus | Totals | %age of Total (133) |
|---|---|---|---|---|---|---|
| VASCULAR | | | | | | |
| Cardiac | 31 | 9 | 8 | 2 | 50 | 37.6 |
| CCF. | 4 | - | - | - | 4 | 3.0 |
| "True" Diab. Nephropathy | - | - | 2 | - | 2 | 1.5 |
| Periph. Vasc. Disease | 1 | - | 3 | - | 4 | 3.0 |
| Cerebral Vasc. Disease | 2 | - | 1 | - | 3 | 2.3 |
| Retinopathy, | | | | | | |
| HPT. | 3 | 2 | - | - | 5 | 3.8 |
| Diabetic | 1 | 4 | 2 | 2 | 9 | 6.8 |
| INFECTIVE | | | | | | |
| Significant TB. | 3 | 2 | 1 | - | 6 | 4.5 |
| UGS Infections | 24 | - | - | - | 24 | 8.0 |
| Various Skin Infections | 16 | 6 | 1 | - | 23 | 17.3 |
| Pruritus Pudendi | 49 | - | - | - | 49 | 36.8 |
| METABOLIC | | | | | | |
| Neuropathy: | | | | | | |
| Subjective | 67 | | - | - | 67 | 51.0 |
| Objective | 5 | | - | - | 5 | 3.8 |
| Cataracts | | | | | | |
| Senile | 24 | | 2 | - | 26 | 19.5 |
| "True" diab. | 7 | 5 | - | - | 12 | 9.0 |
| Liver enlargement | 1 | - | - | - | 1 | 0.8 |
| | (2 CCF) | - | - | (2 CCF) | 4 | 3.0 |
| Diabetic coma | 7 (all adm.in coma) | 2 | - | - | 9 | 6.8 |
| Hypoglycaemic coma | - | 3 | - | - | 3 | 2.3 |

SECTION III - 11 VASCULAR COMPLICATIONS.

In the following table the presence of Vascular Complications at the first examination has been correlated with how long the patient had had diabetes. By far the majority of Vascular Complications, particularly hypertension, were present at the time of onset of the disease.

In this context diabetic nephropathy is regarded as the classical Kimmelstiel-Wilson type of histological picture. Infective Nephropathies are discussed under "Infective Complications".

| | When disease first came on. | 0 - 5 yrs | 5 - 10 yrs. | 10 yrs | TOtal %age, |
|---|---|---|---|---|---|
| Cardiac, including HPT., CCF. (p. 63) — HPT | 31 | 9 | 8 | 2 | 50 37.6% |
| Cardiac, including HPT., CCF. (p. 63) — CCF | 4 | - | - | - | 4 3.0% |
| Nephropathy: (HPT, Retinitis, Albuminuria (p. 67 ) | - | - | 2 | - | 2 1.5% |
| Peripheral Vascular Disease (p. 71 ) | 1 | - | 3 | - | 4 3.0% |
| Cerebral Vascular Disease (p. 66 ) | 2 | - | 1 | - | 3 2.3% |
| Retinopathy: Hypertensive | 3 | 2 | - | - | 5 3.8% |
| Diabetic (p. 70 ) | 1 | 4 | 2 | 2 | 9 6.8% |

SECTION III - 12 HYPERTENSION AND CARDIAC DISEASE.

In all patients, at the first visit, the blood pressure was taken in the left arm by the auscultatory method, using a mercury shygmomanometer, with the patient in the sitting position, after a short rest. If the readings were above the WHO levels of 150/96, then the patient was kept in the sitting position for 20 minutes, and during this time three readings were taken and the lowest accepted as the correct readings. Systolic and diastolic were assessed separately, thus the lowest readings were the lowest systolic and the lowest diastolic recorded in the 20 minutes. This procedure was used to correspond with that used in studying a control group of Zulu people in an urban township (Gampel, B. 1959. Pers. Comm.). In this study the diastolic blood pressure was defined as :- complete disappearance of the sound.

Of the 50 hypertensives, 24 had elevation of both systolic and diastolic, 22 elevation of systolic and 4 elevation of diastolic only.

| | This series. | Controls (Lamontville Location, Durban) (Gampel, B.(1959) Pers. Comm.) |
|---|---|---|
| Age - 25 yrs. | Male 0/1 (-)<br>Female 0/6 (-) | Figures not available.<br>" " " |
| 26 - 35 | Male 0/8 (-)<br>Female 2/13 (13.4%) | 17.7%<br>13.8% |
| 36 - 45 | Male 1/16 (6.3%)<br>FEMALE 10/21 (47.6%) | 15.6%<br>58.6% |
| Over 45 years. | Male 12/25 (40.8%)<br>Female 25/43 (58.1%) | 58.1%<br>60.1% |

Differences between this small series and the controls were not appreciably significant with the exception of the fact that the

SECTION III - 12 (continued)

diabetics appeared to have less in the way of hypertension than the controls. In fifty hypertensives out of the 133 total, significant albuminuria (more than a trace) was seen in 17 (34%): it is not possible to say in the female hypertensives (43) in how many this was due to urinary tract infection.

When first seen, four patients were or had been in congestive cardiac failure. All had or were subsequently satisfactorily treated with Digitalis and Diuretics. The aetiology of all cases appeared to be hypertensive, excepting in one (Case No. 143) where a tuberculotic pericardial effusion was associated with a bundle branch block, who was studied in the ward and was in failure.

Of interest is Case No. 131, a 57 year old male who, since he first attended nine years ago, has been in congestive failure, and who has been on Digitalis and Diuretics all this time. His diabetic control has been very satisfactory on low doses of Tolbutamide (250 mgm. B.D.) and it was believed that he might have been suffering from a mild form of cardiomyopathy.

SECTION III - 12 (continued)

FOLLOW-UP CASES:-

(NOTE: Cases with sudden hypertension and very heavy albuminuria - even without retinopathy, are in the Section on Nephropathy).

Case No. 96 was a 50 year old female who was one of the connubial diabetics in this series, and who was on Insulin for eight years before we saw her. She has attended at the Clinic for nine years and has been very well controlled on Chlorpropamide (125 mgms. B.D.). She developed sudden hypertension of 220/110 in the 17th year of her diabetes, which has required fairly energetic treatment, but has kept well.

Case No. 103 was a 56 year old female who was well controlled on 500 mgms. Tolbutamide T.D.S., but at her last visit was noted to have suddenly developed a B.P. of 240/150. Refusing hospital admission, she was put on to energetic out-patient treatment for this, but did not appear at the Clinic again.

Case No. 121, was a 49 year old female who, in the fourth year of her attendance, was moderately well controlled on 250 mgms. of Chlorpropamide twice daily. At her last visit it was noted that she had developed a heavy albuminuria and a B.P. of 180/110, without diabetic retinopathy. She was put on to energetic out-patient treatment, but was not seen again in the Clinic.

Case No. 40, a 54 year old female, having been poorly controlled on oral treatment following changing from Lente Insulin first to Tolbutamide and subsequently to Chlorpropamide, had a stroke after eight years of attendance and fifteen years of diabetes, preceeded shortly before by a sudden rise of blood pressure of 210/100. She has made a satisfactory recovery, is still attending, but has been put back on to Insulin.

Case No. 149, a 66 year old male, during the second year of attendance and the sixth year of diabetes, went into congestive failure and was satisfactorily treated with Digitalis and oral Diuretic agent.

Case No. 115, a female aged 74, after thee years of very good control on Tolbutamide 250 mgms. T.D.S., went into congestive failure and was satisfactorily treated with Digitalis and oral Diuretic agents.

SECTION III - 12 (continued)

Case No. 150, a 47 year old male, after one and a half years of attendance and seven years of diabetes, developed congestive failure, which was initially easily controlled with digitalis and diuretic agents: however, later he went into irreversible cardiac failure and died. (p. 143)

SECTION III - 13 CEREBRAL VASCULAR DISEASE.

Cerebral vascular accidents are remarkably common in the non-diabetic Zulu, and in Zulus under the age of 40, for some strange reason, constitute the biggest problem of central nervous system disease. This fact is interesting in view of the extreme rarity of clinical coronary artery disease in these people.

In the present series, when first seen at the Clinic, three patients had suffered strokes, Cases Nos. 45, 86 and 161. Cases Nos. 86 and 161 had had their strokes before developing diabetes, and Case No. 45 after.

Case No. 45, Male, aged 58. Eleven years diabetic when first seen. This case was one of the two Kimmelstiel-Wilson disease patients, with early cataracts in addition. About ten years after developing diabetes he had a right-sided hemiparesis from which he made a very good recovery. This patient was also one of the very uncommon Zulu diabetics who had a family history of diabetes, in a sister and a brother. He had been on Insulin for ten years, but was satisfactorily converted to Tolbutamide.

Case No. 86. Female, aged 36. Fat, two years diabetic, well controlled on Tolbutamide, having previously been on Insulin for two years. She had a stroke about one year before developing diabetes. This was a right-sided hemiplegia, and she had made a very good recovery from this. Her blood pressure readings had been normal.

Case No. 161. Male, aged 48 - two years diabetic. He was well controlled on Insulin, 40 units of Lente daily. He had had a stroke a year before becoming diabetic. When first seen he had a B.P. of 170/120, with cardiac enlargement. He suffered a left-sided hemiplegia and was very spastic on that side. Nevertheless, he had made a remarkable recovery, and was back at work as a Schoolmaster.

SECTION III - 13 (continued).

Case No. 86, a 36 year old female, in the second year of her treatment while under moderately good control with Chlorpropamide 250 mgms. B.D., developed a right-sided hemiplegia, without hypertension, from which she recovered fairly satisfactorily. Five months after this, visited the Clinic for the last time and has never been seen again.
Case No. 5, a 45 year old female, in the fifth year of her attendance and her seventh year of her diabetes, developed sudden hypertension while on good control with Tolbutamide (500 mgms. T.D.S.) and sustained a left-sided hemiparesis. She attended for a further two years under good control of her hypertension, having made a satisfactory recovery, and then she disappeared.
Case No. 53, a 49 year old female at present attending the Clinic, is well controlled on Phenformin 50 mgms. time disintegrating capsules B.D., but has developed early signs of hemiparesis with a mild rise in B.P. to 180/110. After two years on no hypertensive treatment, the hemiparesis has now disappeared completely, and subsequently her B.P. has fallen to 150/95.

SECTION III - 14 THE "TRUE" DIABETIC NEPHROPATHY.
(as opposed to the Infective Nephropathies).

The clinical triad of hypertension, albuminuria and retinopathy of the diabetic type that is associated with the classical histological lesion in the kidney of the Kimmelstiel-Wilson type, was seen at first visit in only two patients in this series. (Kimmelstiel, P. and Wilson,C.(1936) Am. J. Path., 12, 83).

In most racial groups this syndrome is generally seen in patients who have been suffering from diabetes for some years (unlike in the Natal Indian, where I have recorded the patient presenting for the first time for treatment with the fully developed syndrome.) In following these two cases, the prognosis does not seem to be as grave as it is in the European patient, where death seems to follow more quickly after diagnosis. In the later stages of the condition, Insulin requirements generally fall, and alleviation

SECTION III - 14 (continued)

has been claimed to follow removal of the pituitary gland, by many workers. In the present series, when first examined, there were two cases - Cases Nos. 30 and 45.

Case No. 30, 51 year old male, diabetes of seven years duration - well controlled on 40 units of Lente Insulin daily. When first seen he had advanced diabetic retinopathy, a blood pressure of 210/110, and a moderate albuminuria. He was in congestive cardiac failure, and this responded well to treatment with Digitalis and Diuretics. This patient also suffered from peripheral vascular disease, and from an objective (and subjective) neuropathy. In spite of all this, he does a hard day's manual labour in a bag factory.

Case No. 45, a 58 year old male, ten years diabetic - had been well controlled on 60 units of Lente Insulin daily for two years. When first seen he had a blood pressure of 220/130, a moderate albuminuria and a diabetic retinopathy which is being obscured by cataracts. In view of the fact that the patient was conscientious, he was given a trial of 500 mg. Tolbutamide thrice daily, and for one year has been very well controlled, without deterioration in his clinical state. His blood pressure is not being well controlled by out-patient hypotensive treatment. There has as yet been no recurrence of a mild left-sided hemiparesis. This patient has two diabetic siblings, a brother and a sister, a most unusual occurrence in the Zulu.

It is interesting to note that in the year 1960, out of eight post-mortems done on diabetic patients (not included in this series) the histological Kimmelstiel-Wilson picture was seen in four of of these patients, and in none of them had the diagnosis been made on clinical grounds, bearing out that the histological renal picture can be found before the classical trial of hypertension, albuminuria and retinopathy are seen.

FOLLOW-UP CASES.

Case No. 2, was a 78 year old female who was well controlled on oral therapy, and in the end was on 125 mg Chlorpropamide B.D. However, at her last visit, it was noted that she had suddenly

SECTION III - 14 (continued)

developed hypertension and a heavy albuminuria, and she was put on to Hypotensive and Diuretic agents, but she was never seen in the Clinic again.

Case No. 30, a 55 year old male, who was well controlled on 80 units of Lente Insulin following a failure of oral therapy, was put back to Insulin but developed diabetic nephropathy in his fourth year of attendance and his eleventh year of diabetes with a hypertension of 170/120, a heavy albuminuria and retinopathy.

Case No. 148, a 48 year old male, developed a heavy albuminuria without retinopathy, but with a sudden hypertension of 180/115, while he was moderately well controlled on 60 units of Lente Insulin and was never seen in the Clinic again.

Case No. 163, a 45 year old male, was poorly controlled on 50 units of Lente Insulin, and in the fifth year of his attendance and the seventh year of his diabetes, developed a heavy albuminuria at his last visit, without retinopathy or hypertension, but was not seen again.

Case No. 87, a 64 year old male, had been on NPH 40 units of Insulin, had had a failure of conversion to Tolbutamide; he was put back on to Insulin again, went into a period of poor control (almost certainly due to not giving himself Insulin regularly); he had a sudden massive weight loss, developed heavy albuminuria with a mild degree of retinopathy and then disappeared from the Clinic.

Case No. 37, was a 61 year old male who, four years after attending the Clinic and in his ninth year of diabetes, was well controlled on 500 mgms. of Tolbutamide three times a day, and developed classical diabetic nephropathy with a B.P. of 200/130, retinopathy and a heavy albuminuria. Following this he was put on to hypotensive and diuretic drugs, but he disappeared from the Clinic.

Case No. 50 was a 69 year old male, who was blind from previous eye infection, and who used to come considerable distances from the country to visit the Clinic. Before he left for the last time, it was noted that his B.P. had gone up to 170/110 and that he had developed a very heavy albuminuria. The retina were not able to be seen well because of his eye disease, but I was fairly sure that he

SECTION III - 14 (continued)

had developed diabetic nephropathy. After this he was never seen again.

Case No. 57, a 54 year old male, after four years' attendance and eleven years diabetes, developed hypertension, a retinopathy and an albuminuria quite quickly over the space of his fifth year of attendance; though he was well controlled on Tolbutamide 250 mgms. T.D.S., and in addition hypotensive and diuretic agents, he disappeared from the Clinic.

Case No. 80, a 52 year old male, who, in the second year of his attendance and in the twelfth year of his diabetes, was noted to have developed a heavy albuminuria without hypertension or retinopathy. Following this he disappeared from the Clinic.

SECTION III - 15 RETINOPATHIES.

An attempt was made to differentiate retinopathies between those who had unmistakable diabetic features (such as microaneurysms and classical aluminium-paint exudates) and those that did not have these features, who were regarded as being hypertensive. "Mixed" with features of both, were regarded as being diabetic. All patients were examined without mydriatics, and if insufficient view of the retinae were obtained, then recourse was, in fact, had to mydriatic agents.

I believe most strongly that opinions on retinae by people who are not trained ophthalmologists, are seldom valid, excepting in the instance of people who had considerable experience in such studies, and in respect of their positive findings only. This has been borne out by an unpublished study on the retinae of one thousand diabetics in my Clinic, where opinions of Ophthalmologist, general physician and registrar have been compared. Thus, what I have set out here are the positive findings obtained by myself in studies carried out as stated above :-

SECTION III - 15 (continued)

Five cases (Nos. 65, 74,103, 123, 131. ) were regarded as being hypertensive retinopathy cases and nine as classical "diabetic" retinopathy. There was no difference between findings in diabetic retinopathy as seen in clinics in temperate climates and what I saw in these patients. In no case, when first seen, did I see proliferative retinitis (or such features as the rete mirabilis) though these have been reported in examination in cases followed up. Blindness due to this cause was not seen.

A striking finding in descriptions of clinical state in patients who subsequently disappeared, was the presence of heavy albuminuria, whether in the presence of hypertension or not. If this presaged a fatal outcome, it appeared likely - but not certain - that severe diabetic nephropathy might occur in the absence of retinopathy.

FOLLOW-UP CASES.

Case No. 151, a 42 year old female, developed severe diabetic retinopathy during the six months she was treated for tuberculosis in the Chest Hospital.

Case No. 118, was a 38 year old female, who is still attending and who developed a glaucoma due to the fact that she did not attend the hospital regularly enough, as a result of acute "true"diabetic cataracts: following attempted treatment of these, she was found to have as well as a glaucoma, a marked degree of proliferative retinitis which may have ante-dated the development of the acute cataract.

SECTION III - 16 PERIPHERAL VASCULAR DISEASE.

Peripheral vascular disease is one of the less common complications of diabetes in the Zulu patient. Coldness of the feet is a very common complaint, but this is not regarded as being evidence of vascular disease. The following criteria were established and patients having one or both of these were considered to be suffering from peripheral vascular disease :-

(1) Classical intermittent claudication, whatever the state of

SECTION III - 16 (continued)

the peripheral pulses.

(2) Coldness of the feet, absence of all peripheral pulses (distal to the popliteal, that is to say, posterior tibial, dorsalis pedis and perforating peroneal pulses), with or without trophic changes in the skin of the toes and preferably without evidence of objective diabetic neuropathy, though the syndromes of peripheral vascular disease and objective neuropathy are often mixed.

Four patients came into this category - Cases Nos. 30, 34, 80 and 125.

Case No. 30. Male, 51 years, thin, seven-year diabetic. B.P.220/110. Kimmelstiel-Wilson's syndrome. The complaint of classical intermittent claudication, was found to have very cold feet, with posterior tibials only just palpable. No trophic changes present.

Case No. 34, Female, aged 63, thin, three-year diabetic. B.P. 200/100. She had been on good control on 1,500 mg. Tolbutamide daily, after having had 20 units Lente Insulin daily for two years. She complained of pains and coldness in the feet. Popliteal and foot pulses were found to be absent with trophic changes in the skin of all toes and slight superficial fungus infection. There was no evidence of neuropathy.

Case No. 80, Male, aged 50, fat, ten-year diabetic. B.P. 170/95. Good control on 1,500 mg. Tolbutamide daily, having had 40 - 60 units Insulin daily for ten years. In spite of easily palpable peripheral pulses, he complained of severe intermittent claudication, the "distance" being at about 100 yards. This did not improve with treatment.

Case No. 125, Male, aged 44, thin, ten-year diabetic. He had fair control on 40 units Insulin daily. Sepsis of two toes on the right foot was associated with loss of all peripheral pulses, without history of claudication. The pulses in the left leg were palpable but faint. The sepsis responded well to anti-biotic therapy, but pulses were still impalpable at the end of treatment.

The duration of the disease in these cases:-3, 7, 10 and 10 years. All of them were hypertensive, and the control in three of them had been fairly satisfactory.

SECTION III - 16 (continued)

Amputation for peripheral vascular disease in the Zulu diabetic is extremely rare, as opposed to the Natal Indian and on whom it is done very commonly indeed. Subsequently I have seen amputation performed on a 70-year Zulu diabetic in the Renishaw Hospital, Natal South Coast, for a popliteal thrombosis of the left leg of slow onset. This appears to be a rare operation in the Zulu. Further, the Professor of Music at the University College of Zululand, a mild diabetic, has subsequently had both legs removed below the knees because of simultaneous bilateral gangrene after ten years of diabetes. In this case, angiography ruled out aortic saddle disease or disease of the femoral vessels.

SECTION III - 17 INFECTIVE DIABETIC COMPLICATIONS.

Most of the "infective" complications were seen in the early stages of the disease. Active tuberculosis was seen in only six out of the 133 patients, at the first visit, which is an apparently surprisingly low incidence, as compared to the general Zulu population, and testifies to the fact that our patients were probably better housed and fed than the general population.

INFECTIVE COMPLICATIONS.

| | When disease first came on | 0 - 5 yrs. | 5 - 10 yrs. | TOTAL % age. |
|---|---|---|---|---|
| Active Tuberculosis at any stage of the disease or before (p. 75 ) | 3 | 2 | 1 | 6 4.5% |
| Urinary Tract infections (p. 78) | 24 | - | - | 24 18.0% |
| Skin Infections including breast abscesses etc. and boils (p. 79 ) | 16 | 6 | 1 | 23 17.3% |
| Pruritus Vulvi and Pudendi. (p.79 ) | 49 | - | - | 49 36.8% |

SECTION III - 17 (continued)

Cholecystitis is virtually unknown in the Zulu. No cases were seen in the present series of diabetes.

General Infections reported in follow-up cases:-

Case No. 66, was a 60 year old female who, in the second year of her attendance and the eighth year of her diabetes, had been changed satisfactorily after six years on Insulin to Tolbutamide with very good control. However, she developed severe pneumonia following a cold, and was admitted into hospital, in extremis, in diabetic coma and died. a Post-mortem showed a fulminating broncho-pneumonia.

Case No. 153, was a 49 year old female, who developed a severe left sided Quinsy requiring operation under general anaesthetic for this, and she was converted from a good control with 500 mgms. of Tolbutamide T.D.S. to soluble Insulin: subsequent to the operation she was put back on to oral treatment again on 250 mgms. Tolbutamide B.D. with moderately good control.

Case No. 6, was a 39 year old female who, during her period of good control on Chlorpropamide ½ tablet B.D., developed severe Herpes Zoster of one of the thoracic nerves, which did not affect the treatment of her diabetes, and from which she made a spontaneous recovery and remained on oral treatment.

Case No. 167, a 56 year old female, was another subject who developed a severe Herpes Zoster in a thoracic nerve, but this did not influence good diabetic control, on one timed desintegrating capsule of Phenformin twice daily.

Case No. 29, a 36 year old female, developed salpingitis, which, for a short while, resulted in very poor oral control of her diabetes on oral therapy and necessitated her being put back on to Insulin for a short while. Following this she was put back on to Tolbutamide 250 mgms. T.D.S. again with good control.

SECTION III - 18 TUBERCULOSIS

It has been known for hundreds of years that diabetics are notoriously prone to develop tuberculosis. Bearing this fact in mind, the procedure in my Clinic is that no matter how soon before the patient may have had his chest X-rayed in routine screening surveys, it is done again at the first Clinic visit and six-monthly thereafter. In the present series at first visit it was found that three patients (Cases Nos. 114,145,169 ) had evidence of previous infection, when the disease came on; two had developed TB between onset and five years later, and one had developed TB five years after onset of diabetes. Here I define "tuberculosis" as a tuberculous infection necessitating formal treatment, and I do not include cases found on X-ray to have minimal scarring or calcification.

All these patients had been treated on drug therapy, chiefly INAH in very large doses (up to 15 mg. per kg. daily!) with or without streptomycin being given them. No surgery had been performed in any case, and all these patients had responded well to treatment of the TB coincidence with satisfactory control of their diabetes.

In view of the rather unusual use of huge doses of INAH by the local TB authorities in Durban, namely up to 10-15 mg/kg per day, in the King Edward VIII Hospital, we see considerable numbers of cases of INAH toxicity, and distinguish four varieties :-
(1) Maniacal (2) peripheral neuritic (3) Myelitic and
(4) a pseudo-muscular-dystrophy type.

Paradoxically, in the six Zulu tuberculous diabetics at first visit no signs of INAH toxicity were seen, even though patients were on doses of the order mentioned above, and even though the Zulu diabetic is particularly liable to subjective diabetic neuropathy.

Speaking now from subsequent experience of large numbers of TB Zulu diabetics, I advise categorically that, excepting in the very mildest cases, patients should be changed from oral therapy to Insulin.

It has been suggested in "mild" cases,(I refer to those whose post-prandial blood glucose levels at one hour do not exceed 200 mg. % on oral therapy - single or combined) that INAH, even in moderate

SECTION III - 18 (continued)

dosage, may restrict pancreatic Insulin productions.
It is wise in these cases to assume this and rely more upon Insulin, as in poorly controlled Zulu diabetics, Tuberculosis may be fulminant.

FOLLOW-UP CASES.

Case No. 82, a 53 year old female in the second year of her attendance and the fourth year of her diabetes, developed a classical INAH neuritis on treatment for active Tuberculosis while being well controlled with Tolbutamide 250 mgms. T.D.S. She did very well indeed but at her last visit at the Clinic, it was noted that she had developed a heavy albuminuria without Ketones or retinopathy, and she was never seen again in the Clinic.
Case No. 146, was a 46 year old female, who was well controlled on 80 units of Lente Insulin; in view of her overweight she was put on to Tolbutamide 500 mgms. T.D.S. with apparent profit. She suddenly developed weight loss and chest signs, and was shown to have active tuberculosis, and she was put back on to Insulin again during eight months in the Chest Hospital. She recovered completely and now is still attending on 20 units of Lente Insulin a day.
Case No. 151, a 42 year old female, was poorly controlled on Tolbutamide when first referred to the Clinic. She was changed to Lente Insulin 40 units a day, on which her control proved even to be poorer, owing to lack of insight into the necessity for regular injections and ignorance of syringe hygiene. During this time she developed acute tuberculosis which required admission to the Chest Hospital after four years duration of diabetes. During her treatment in the Chest Hospital, over six months, she developed marked diabetic retinopathy. When she attended the Clinic after discharge from the Hospital, she was kept on Insulin. However, subsequent to this she disappeared and has not been seen again since.
Case No. 162, a 55 year old female, after attending for two years, had a sudden enormous weight loss with cough and expectoration, when moderately well controlled on Tolbutamide 500 mgms. T.D.S, and referred to the Chest Hospital where she was found to be tuberculous, but was never seen again at the Clinic.

SECTION III - 18 (continued)

Case No. 111, a 64 year old female, went into a sudden poor control whilst on Tolbutamide, developed severe acute tuberculosis and died in the Chest Hospital.

Case No. 63, a 49 year old male, in whom conversion to oral therapy from Insulin was apparently satisfactory. He went into a period of very poor control during or prior to an attack of acute tuberculosis, and had to be put back on to Insulin, but made a good recovery after five months in the Chest Hospital. He is much better and is still attending, on 50 units of Lente Insulin a day.

Case No. 48, a 51 year old obese female, who in the third year of her attendance at the Clinic and fifth year of diabetes, developed tuberculosis which came on during a period of bad control with oral therapy on Tolbutamide 500 mgms. T.D.S. She was changed to 60 units of Lente Insulin a day in the Chest Hospital, and has done very well since. After seven months she has been discharged as completely fit, still on Insulin.

Case No. 24, a 61 year old obese female developed tuberculosis whilst on apparently good control with Tolbutamide 500 mgms. T.D.S. Soon after this she disappeared from the Clinic, having been referred to the Chest Hospital.

OTHER RESPIRATORY CONDITIONS.

Case No. 154, a 46 year old male, was well controlled on Chlorpropamide 125 mgms. daily, and developed bronchial asthma, which required admission to hospital. This is an uncommon condition in the Zulu.

Case No. 9, a 67 year old male, who after two years of attendance and ten years of diabetes, developed respiratory symptoms and large hilar glands on chest X-ray. In consultation with the Chest Physicians, it was decided not to treat him but to watch him. The hilar glands disappeared without any treatment whatsoever while the patient was well controlled on 250 mgms. of Tolbutamide three times a day.

Case No. 38, was a 56 year old female who was on good control with Tolbutamide 250 mgms. B.D. having originally been on 1000 mgms. T.D.S. However, her son was murdered, and she developed a psychological shock and severe bronchial asthma, and then disappeared from the Clinic.

## SECTION III - 19 THE INFECTIVE NEPHROPATHIES, AND URINARY TRACT INFECTION.

Urinary tract infections are extremely common in the middle-aged Zulu female, whether diabetic or not, and I have gone so far on the basis of subsequent studies of the urine of such patients, to say that I do not believe that urinary tract infection is ever absent in the lower class non-white diabetic female. Predisposing causes are poor pudendal hygiene, aggravated by local obesity and profuse sweating.

In this series urinary tract infection was said to be present if the patient complained of marked dysuria, frequency and superpubic discomfort, or offensiveness of the urine. These symptoms were complained of by 24 out of the present series of all women on their first visit, and in none of them was loin pain felt, suggesting that the infection, in all probability, was confined to the bladder without having ascended to the kidneys. In no case were urinary symptoms regarded as being severe enough to warrant further urological investigation, and most cases responded readily to diabetic control, the use of Mandelamine, the prescription of a dusting powder, vaginal pessaries, and advice about pudendal cleanliness. I have found that it is useless to attempt treating urinary tract infection in the female Zulu diabetic without using vaginal pessaries, preferably of the "Mycostatin" or anti-fungal type.

Clinical assessment about the clearance of urinary tract infections is unreliable , and, in cases where there was any doubt, Mandelamine was used in long term therapy (1 tablet T.D.S.) as a drug of choice-being effective, cheap, and not conducive of resistent B. coli.

In all the ten cases on whom urine cultures were done in the wards, the infective organism was found to be B. coli. The absence of faecal streptococci was quite striking.

As regards the males, no cases of prostatitis or urinary tract infection was seen, though prostatic enlargement was found in a single case (Case No. 133), operation not being considered necessary.

SECTION III - 19 (continued)

FOLLOW-UP CASES.

Case No. 62, a 40 year old female, has developed repeated urinary tract infection and has been admitted to Hospital on numerous occasions for special studies, which have shown, on most occasions, that the infecting organism has been a B. coli. Her diabetes has remained reasonably well controlled during this time on 250 mgms. Tolbutamide B.D.

Case No. 134, was a 52 year old female, still attending, who was converted well from Lente Insulin 40 units, to 500 mgms. and subsequently 250 mgms. of Tolbutamide T.D.S. She has had persistent mild urinary tract infections, for which specialised investigations have not revealed the cause. She is now kept on long term Mandelamine.

SECTION III - 20 PRURITUS PUDENDI.

Pruritus pudendi in this series, as in other series of diabetics, was extremely common, and was seen in exactly half the females, and in 4% of the males.

The relationship between pudendal infection and bladder infection is well known, and has been noted on p. 78 : therapeutic measures were set out as being frequent bathes, dusting powder, mycostatin pessaries during relapses, and long term urinary antiseptic such as Mandelemine.

SECTION III - 21 BOILS, SUPERFICIAL AND DEEP ABSCESSES.

(A) BOILS: These were recorded in ten patients at their first visits (8 males and 2 females). They were commonest on the back of the neck, but in the two females they occurred in the pudendal region. They resulted in very little upset in diabetic control, indeed, in four patients, two males and two females, control had been satisfactory on the oral agent - Tolbutamide.

SECTION III - 21 (continued)

FOLLOW-UP CASES.

Case No. 175, a 41 year old female, was poorly controlled on Tolbutamide 500 mgms. TDS, and was changed to Acetohexamide 500 mgms. TDS with considerable improvement. During the time after this conversion, she developed severe crops of boils, chiefly due to very poor body hygiene. Strange to relate, these boils did not affect the standard of diabetic control. Large doses of depot penicillin (Bicillin) were given with good results. However, following a period of good control subsequently, she disappeared from the Clinic.

It is very important to note the remarkable absence of skin infections after the Clinic was started. The Consultant in Dermatology at the Hospital (Leeming, J. (1959) Pers. Comm.) said that with the starting of the Diabetic Clinic and especially with the wholesale use of the oral anti-diabetic agents, the numbers of diabetics attending his Clinic had diminished drastically.

(b) BREAST ABSCESSES:

These were recorded as having been seen in five parous women (Cases Nos. 3, 25, 82, 100 and 123). Unfortunately no attempt had been made to stop these women lactating after delivery and this is an extremely important factor in the prevention of breast abscesses in people who are not always clean and whose control of diabetes might not be satisfactory. All these cases required surgical intervention, and the abscesses were present in all cases, only in one breast. The commonest organism being recorded was a Staphylococcus pyogenes.

DEEP INJECTION ABSCESSES.

Large and severe injection abscesses were seen in two cases. Case No. 26 lapsed into diabetic coma when she developed a large abscess, and Case No. 142 required wide surgical resection for a huge gluteal abscess caused by an infected insulin injection site.

This patient was very fat, but was unusual in being truly dependent upon insulin, attempts to convert her to oral therapy in the Wards being without avail. Indeed, finally(see p. 95), she died in diabetic coma in the Hospital.

SECTION III - 21 (continued)

PALMAR SPACE INFECTION.

A 44 year old male (Case No. 148) developed a severe palmar space infection whilst on Insulin therapy. which required admission to surgical wards and incision. He has remained well controlled on 60 units of Lente Insulin daily since then.

Of the total series of 133 diabetics only 18 had had at any time boils or abscesses - a remarkably low incidence, when one considers that their standards of cleanliness are not as high as one might find in more advanced countries.

FOLLOW-UP CASES.

Case No. 175, a 41 year old female, who was poorly controlled on Tolbutamide 500 mgms. T.D.S. and was changed to Aceto-Hexamide one tablet T.D.S. with considerable improvement. However, during this time she had started severe crops of boils, chiefly due to very poor bodily hygiene, but, strange to relate, these boils did not result in any deterioration of her clinical diabetic state, and were treated with large doses of Penicillin. However, following a period of very good control she disappeared from the Clinic.

SECTION III - 22

THE TABLE OF INCIDENCE OF METABOLIC COMPLICATIONS

The diabetic complications seen in these series that can be regarded as "Metabolic", as opposed to "Vascular" or "Infective, include the following :-

(1) Subjective diabetic neuropathy.
(2) Objective diabetic neuropathy.
(3) The "senile" diabetic cataract.
(4) The "true" diabetic cataract of young people.
(5) Liver enlargement.
(6) Diabetic coma.
(7) Hypoglycaemic attacks.

The following table shows the number of patients in the total series who developed these complications and the approximate duration of the disease before these became apparent. In respect of the senile cataract and the neuropathies, where it is difficult to establish, when in the disease, they first appeared, the categories of 0 - 5 year diabetics and the new diabetics were grouped together.

No cases of xanthomata disseminata, xanthoma tuberosum or necrobiosis lipoidica diabeticorum were seen in these series; nor even in spite of a special interest in necrobiosis lipoidica have I noted a single case in the very large total of non-white diabetics that have been registered in our Clinic for the last nine years.

SECTION III - 22

TABLE SHOWING NUMBER OF DIABETICS WITH "METABOLIC" COMPLICATIONS, AND RELATIONSHIP WITH APPARENT DURATION OF THE DISEASE(133 Pts.)

| | When disease first came on. 0 - 5 years | 5 - 10 years | 10 years or more. | TOTAL and %age. |
|---|---|---|---|---|
| Subjective neuropathy (p. 84) | 67 | - | - | 67 (51.0%) |
| Objective neuropathy (p. 85) | 5 | - | - | 5 (3.8%) |
| "Senile" cataract (p. 87) | 24 | 2 | - | 26 (19.5%) |
| "True" Diabetic Cataract (p. 89) | 7 5 | - | - | 12 (9.00%) |
| Liver Enlargement (p. 92) | 1<br>2 CCF - | - | 2 CCF | 1 (0.8%)<br>4 (3.0%) |
| Diabetic Coma (p. 92) | 7 ++ 2 | - | - | 9 (6.8%) |
| Hypoglycaemic Coma (p. 96) | 3 | - | - | 3 (2.3%) |

++ admitted in coma.

NOTE: "Subjective Neuropathy" = any abnormal uncomfortable sensation in the legs, that is relieved by treatment.

"Objective Neuropathy" = include cases who have loss of vibration sense, or actual local loss of sensation to pin-prick

SECTION III - 22 (continued)

There was only one case of local lipoatrophy to Insulin in this series and this was :-

Case No. 49, a 22 year old female who, after 5 years of attendance, developed quite a severe local lipoatrophy due to her Lente Insulin progressing even though she was changed to NPH insulin. She announced that she was getting married, was given a good supply of Insulin but, unfortunately, was never again seen in the Clinic.

General Diseases:
Case No. 169, a 52 year old male, was excellently controlled on Tolbutamide 500 mgms. T.D.S. dropping to 250 T.D.S. He developed mild rheumatoid arthritis, which is an extremely rare syndrome in a Zulu.

SECTION III - 23.

SUBJECTIVE DIABETIC NEUROPATHY.

I define "subjective diabetic neuropathy" in the Zulu diabetic as (1) any abnormal uncomfortable sensation in the legs, at the onset of diabetes, that is relieved by treatment, or (2) similar sensations coming on later in the syndrome which are not always affected by treatment but which seem to be self-limiting. Between these two there seems to be a hiatus of time when one does not experience these symptoms. If there are abnormalities of vibration sense, or sensation to pin-prick, the patient is regarded as a case of "objective neuropathy".

Subjective diabetic neuropathy was the commonest presenting symptom in the present series, being complained of by 50% of patients.

The above rather comprehensive definition of "subjective neuropathy" was coined because of the fact that the Zulu word "Inkwantshu" - invariably used to describe the symptoms, indicates in Zulu "any abnormal subjective sensory symptom". Diabetic neuropathy is unfortunately hardly explained by the words "Kuyahlaba" - pricking:

SECTION III - 23 (continued)

"Kuyashisa" - burning: (with the exception of the nocturnal symptoms by some patients, but which are invariably referred to as "Inkwantshu") or "Isidubuli" or shooting, or "Kuyabamba" - cramplike.

As stated above, the syndrome in the new diabetic generally goes away with the institution of treatment; in the established diabetic I believe that these symptoms are self-limiting and not really affected by treatment. Attempts to treat these symptoms with Vitamin B.12, are generally fruitless though in a few cases recently, not includedin this series, remarkable relief has been obtained by the use of Phenytoin.

Case No. 45 was a 61 year old obese female, who was poorly controlled on 40 units of Lente Insulin and was changed to Tolbutamide with very satisfactory control indeed and weight loss. On many occasions she developed mild subjective neuropathy for short periods for no reason whatsoever, which disappeared without any special treatment.

SECTION III - 24.

OBJECTIVE NEUROPATHY.

Objective Neuropathy covers the syndromes of nerves involvement in diabetes where abnormalities were demonstrable by clinical examination as opposed to patients suffering from purely subjective neuropathy. In many normal Zulus deep tendon reflexes are absent. Thus I regard absense of vibration sense or sensation of pin-prick in local areas as being the cardinal signs.

No cases of optic atrophy or Argyll-Robertson pupil were encountered. A case who had a unilateral apparent Argyll Robertson pupil was in fact shown to be suffering from the late effects of a posterior synechia. The pseudo-tabetic bladder was not seen in this series or in a subsequent series of many hundred patients.

If the definition of subjective neuropathy, as stated above, includes the words "relieved by treatment", or "self-limiting",

SECTION III - 24 (continued)

then sustained impotence, probably being due to a visceral neuropathy, must come into the category of objective neuropathy. This is the most distressing and common complication of diabetes in the male Zulu, for it is seldom amenable to therapy, and loss of potency amongst these people brings on a most distressing social stigma. No less than 66% of the males in the present series presented with this condition and in only a few of these did the treatment of diabetes have any effects whatsoever. In ten cases a combination injection of gonadotrophic and male sex hormones "Testotropin" apparently had some effect on erection, but it is difficult to know whether this was due to the actual hormone or to a placebo effect.

None of the unusual diabetic syndromes, such as myelopathy, were seen. Although a muscle wasting was seen in two patients with tuberculosis treated by the local regime of large doses of INAH, it is difficult to know whether this was in fact due to that drug or to the synergistic action of a mild diabetic neuropathy.

In this category I have not included conditions such as cerebral vascular accidents. It is interesting to note, though occasionally seen in Indians, the cranial nerve palsies sometimes associated with diabetes, were not seen in the present case of Zulu diabetics.

Follow - up cases:-

Case No. 85 was a 44 year old male, on good control with Phenformin 50 mgms. (delayed acting capsules) one B.D., has developed an objective neuropathy with loss of vibration sense in both ankles and has areas of anaesthesia to pin-prick in his toes. At the same time, he has become totally impotent and has developed moderate albuminuria without hypertension or retinopathy.

SECTION III - 25

## THE SENILE DIABETIC CATARACT.

There was a disproportionate number of patients in this series suffering from cataract. This is almost certainly because cataract is the chief surgical cause for diabetics being referred to a hospital dealing with Zulu or Indian patients. The most striking feature was the number of patients suffering from the "true" diabetic cataract of young people. Altogether of the 133 patients, no less than 38 patients were suffering from cataract and 26 of these (21 females and 5 males) were considered to belong to the "senile" cataract group, and the main differences and possible dividing cataracts are set out in a table in the next section.

Of the 26 in the senile group, the vast majority (23) came on slowly and in every way their syndrome was so similar to that seen in any European diabetic Clinic. In three cases the development was very quick, and in each was seen the "snowstorm" picture with the slit-lamp microscope that is generally believed to be cataract of the "true" diabetic cataract, but which I feel confident is a measure, in any age group, of cataract developing in patients with wide Blood Sugar swings.

Here, I must impress my indebtedness to the surgeons in the Eye Department, who taught me how to use the slit-lamp microscope and allowed me access to this invaluable and beautiful instrument.

All patients with "senile" cataract were operated on one side alone, as far as followed, and of the 26, results were satisfactory in 16, and in 8 there were less than satisfactory. In 2 patients, eyes were lost.

I believe that poor results are chiefly due to bleeding on the 4th or 5th day - mainly a manifestation of further wide Blood Sugar swings, - and to cross infections in the hospital wards. Of the most important things to ensure success, preventing wild fluctuations of Blood Sugar levels in the first 10 days, I believe

SECTION III - 25 (continued)

to be paramount.

This is very difficult in Zulu patients on oral therapy, and I advise that all operations should be done only after the patient has been put on to Lente Insulin; I find that there is generally no necessity for a full week in a medical ward to do this, chiefly as beds are too precious in this hospital. I believe that a single full pre-operative day on Insulin is normally enough. In very busy wards, it is safer to rely on the once daily Lente Insulin then twice or thrice daily soluble Insulin, which might possibly be better in flattening wide Blood Sugar swings. In the Zulu, urine sugars parallel blood sugars more satisfactorily than in the Indians for instance, and for practical purposes suffice - rather than having to do a repeated random blood sugar levels.

In all cataract cases in the Zulu people one should lean towards the more radical attitude because of the possibility of poor clinic attendance, such as in one case where glaucoma intervened on both sides causing blindness in a cataractous young woman who had stretched out her attendances a little too far. Thus it is interesting to read the leader on "Diabetes & Cataracts" BMJ (i, 934) of 16th April, 1966, where it was stated that good control may obviate the need for waiting for the cataract to mature.

Follow-up Cases.

Case No. 35 was a 65 year old male, who was converted from 40 units of Lente Insulin to good diabetic control on 125 mgms. of Chlorpropamide twice daily. After attending the hospital for 9 years and being diabetic for 14, he developed classical senile cataracts of diabetes which have been subsequently operated on with success.

SECTION III - 26.

## THE "TRUE" DIABETIC CATARACT OF YOUNG PEOPLE.

The most striking feature in this survey of Zulu diabetics was the commoness of the "true" diabetic cataract of young people, indeed in the first month after starting the Clinic, no less than three of these patients were seen; I was so impressed by this, that I arranged a special meeting to demonstrate these cases, as being what would be ordinarily an extremely rare syndrome in Clinics in Europe or America. Interestingly enough, within the next six months a further 9 cases were seen.

In view of the fact that cataracts were seen over almost the whole age range of my diabetic patients, it appeared essential to try and fix an upper arbitrary age limit of patients, under which cataracts were more characteristic of the so-called "true" diabetic cataract of young people. In Clinics, in more temperate climates, this cataract is generally defined as a fast-maturing cataract sometimes reaching maturity in a few weeks, invariably bilateral, coming on in younger patients in whom diabetic control is almost always unsatisfactory, and in whom the cataract is believed to be due to acute coagulation of lens protein. Using the slit-lamp microscope to follow these more acute cases, one sees the characteristic "snowstorm" pattern which coalesces very quickly into the matured cataract.

In the present series, though the "snowstorm" pictures were occasionally seen in elderly patients developing cataracts very quickly, the criteria, as stated above, were most commonly fulfilled by patients under the age of 45 years of age, and so I believe that this age can be regarded as being the upper age of onset of the "true" diabetic cataract. No less than twelve out of the total of 133 Zulu diabetics suffering from this syndrome, which must be regarded as an extremely high incidence, but once again, can be explained on the basis of the commonest of cataract as a surgical diabetic problem.

SECTION III - 26 (continued)

It was of interest that in 1959 a Registrar arrived from the Moorfields Hospital in London, where, in the course of some years study, he had never seen a case of a "true" diabetic cataract, but in the space of his first 3 weeks in our Hospital, I was able to show him what he referred to as "true" cataracts at the "staggering rate" of one a week.

These cataracts cause great disability; they occur most commonly in young women who have families to raise and can render them temporarily blind in the space of a few months, even if they do not get glaucoma. Surgical removal is far from satisfactory, as the Eye Surgeons themselves admit, and frought with complications - mostly haemorrhagic, which lead to loss of the eye.

In this series of 12 cases, 8 were in females and 4 in males, and in all cases operations were done on the one side. These were successful in 7 cases, and in 3 women and 2 men complete failures, resulting in loss of vision in the eye concerned. Of these failures, 4 were subsequently operated on the other side, fortunately with success, and almost certainly due to the fact that during the first 10 days following operation, the patients were kept almost continuously hypoglycaemic with liberal doses of Lente Insulin, and on as strict a low carbohydrate diet as was possible.

In an attempt to summarise the differences which I have observed, and to set out what I believe to be a reasonable age limit to the cases of "true" diabetic cataracts, I append the following table :-

SECTION III - 26 (continued)

TABLE SUMMARISING SUGGESTED SUBDIVISION OF CASES OF DIABETIC CATARACTS.

| | "TRUE" DIABETIC CATARACT | SENILE DIABETIC CATARACT |
|---|---|---|
| Suggested Age Subdivision. | Up to 45 years | 45 years and over. |
| Method of onset | Often very rapid. May be slow in some cases. | Generally very slow over months or years. |
| Type of Diabetic | Often labile Insulin-dependant diabetic with swings in Blood Sugars. Marked Blood-Sugar swings are invariable in the extremely acute cases. | Generally not labile diabetics, but people on oral therapy. No marked swings in Blood Sugars. Even if Blood Sugars stay at high levels they do not swing. |
| Sex Ratio | 4 male 8 female (12) | 5 male 21 female (26) |
| Slit-lamp appearance | "Snowstorm" common, but not always present. Chiefly seen in labile patients. | "Snowstorm" very uncommon but it is seen. |
| Acute cases | 10 out of 12 | 3 out of 26 |
| Complications | Glaucoma occasionally. 1 out of 12 cases. | Glaucoma not seen in this series. |
| Operative Results | Not good. Difficult to keep Blood Sugars "flattened." | Better than in the more acute case. In this series "good" in 16/25. |
| In this Series 38 cases out of a total of 133 | 12 cases | 26 cases. |

## SECTION III - 27 LIVER ENLARGEMENT.

The pathology of liver disease in the Zulu diabetic is discussed on page 142. Suffice it is to say that in the present series of patients, five were recorded as having had significant clinical enlargement of the liver. Of these, this enlargement was considered due to heart failure in 4, responding to conventional treatment with Digitalis and Diuretics. In the fifth case (No. 116), a senile or "peasant" diabetic, a female of 74 years of age who had just become diabetic, there was a liver which was hard, though smooth, the edge of which was palpable between 4 - 5 cms. before the right costal margin. Biopsy was performed on this patient, and this showed a marked degree of siderosis (an unusual finding in our diabetics). It is interesting to note that, unlike European haemochromatolics, this patient's diabetes was controlled only with very great difficulty, daily doses of Insulin in excess of 100 units, resulting in variable and unsatisfactory control.

It should be noted that the extreme obesity of many of my patients precluded accurate localisation of the liver edge. In no patients was an enlarged spleen found. Strangely enough, liver enlargement in patients with ketosis and coma, due to acute fatty infiltration (so common in temperate diabetics) was conspicuous in its absence.

## SECTION III - 28 DIABETIC COMA AND KETOSIS.

In the Zulu diabetic, especially in the earlier series where there was a larger incidence of Insulin-dependent patients than have been encountered in more recent years, ketosis and coma were very common, mostly on the grounds of forgetting Insulin, or running out of Insulim, or when huge injection abscesses occurred. No less than nine of the present series of patients were admitted at one time or another in coma. (Cases: 46, 66, 82, 98, 126, 142, 147, 170 and 174.) Zulu diabetics admitted in coma are generally desperately ill because it is a common thing for friends and relations in any illness invariably to adopt a "wait and see" attitude; even when they decided that the patient should be

SECTION III - 28 (continued)

referred to hospital, there is often a considerable time lag and the patient is generally dramatically ill, and energetic and accurate treatment is required at once. Before the present series was written up, what records I was able to find show that in our Hospital diabetic coma was almost invariably fatal. The following regime has been worked out, and in the last twenty patients admitted, who have been thus treated, only four have died.

It is most important to remember that treatment of this condition should not be delayed until the patient goes to the ward, and that all out-patient departments should have a liberal supply of soluble Insulin which should be given at once, and I believe that the best intravenous infusion to give initially is plasma, in an attempt to restore the circulating blood volume to the brain, kidneys and the adrenal gland, as these patients are often in the ultimate stages of medical shock. Thus, the regime I advise can be summarised as follows :-

(A) Immediately on arrival in the out-patient department, 200 units of soluble Insulin should be given intravenously.

(B) Two bottles of plasma are run into an arm vein as quickly as possible. If this is not possible, due to collapsed veins, it should be run into the femoral vein without delay, which is a much easier procedure. If possible, a blood sugar should be estimated by the use of Dextrostix.

(C) A maximum of three litres (in large patients) and a minimum of two litres (in small patients) of normal saline are run into an arm vein and if these are still collapsed, into the femoral vein, as quickly as possible. Only after this should the patient be sent to the ward. It is very important in such patients not to exceed three litres of saline in the first 24 hours as they appear to develop congestion of the lungs very quickly indeed.

(D) As soon as the saline has been run in, the femoral drip is discontinued, and if an arm drip is working, 5% Dextrose in water is run in now as the sole intravenous regime in

SECTION III - 28 (continued)

the next 24 hours, (the rate depending upon the clinical degree of dehydration).

(E) In spite of what is advised, in more civilised clinics, a catheter is inserted into the bladder and hourly urines are taken off. A broad spectrum antibiotic is prescribed to combat urinary tract infection.

(F) The use of 1/6 molar lactate may prove advantageous when ketotic breathing is marked. It is wise to run in a 150 ml. vial of this into the same drip as the Dextrose is running through. As soon as the patient has recovered enough to swallow, sodium bicarbonate and potassium citrate are given by mouth (50 ccs. of each of the standard solution). If this recovery is delayed, it is essential that a Ryles tube should be passed and that these substances should be run into the stomach.

(G) Vomiting is not apparently a conspicuous feature in these dreadfully ill Zulu patients, and we have not encountered the acute dilatation of the stomach that is described from temperate climates. However, if the patient is vomiting, a large stomach tube should be passed without delay and the contents of the stomach washed out. Subsequent to this, potassium citrate or a bicarbonate solution can be installed into the stomach before the tube is withdrawn.

It is interesting to note that with the use of these enormous doses of Insulin (which should be repeated after an hour if there is no improvement, and hourly after that), that the changes in Blood Sugar levels are not apparently affected by the Dextrose Infusion.

(Dextrostix Ames) has proved invaluable in signalling to the doctor in charge any rapid falls in Blood Sugar, and these finger prick estimations should be done half hourly. When Blood Sugar starts to fall in very ill patients, potassium administration is obligatory, and if by this stage the patient has been unable to swallow any, a falling Blood Sugar should be an urgent sign that the Ryles tube should have been passed and the potassium citrate and sodium bicarbonate instilled into the stomach. In these

SECTION III - 28 (continued)

dramatically ill patients, I believe that it is unwise to give potassium intravenously, and that the gastric route is the route of choice.

Two cases are quoted as being of interest :-

Case No. 147: A 22 year old female diabetic weighing 120 lbs, was admitted three times in severe coma. On the second occasion she was given no less than THIRTEEN LITRES of fluid in the first 24 hours, and altogether 1500 units of soluble Insulin, 600 of which were given intravenously. The rule of "no more than 3 litres of saline" in the first 24 hours was strictly observed, and she did very well indeed. On her third and last admission, her coma was clinically not worse or of longer duration, but during her treatment, which was conducted on similar lines to the above, she was treated by a registrar who did not adhere to the "three litres of saline" rule, and she received six litres of saline in the first 24 hours, and died suddenly after this.

Case No. 142: Another patient, female aged 18, was admitted in coma with a blood sugar of 1800 mg%, and she apparently was improving, until by mistake a vacoliter of 5% saline (instead of 5% glucose) was run in. She died during this infusion in spite of the fact that her serum potassium was, at the time of death, within normal limits, and the Blood Sugar had fallen from 1800 to 1000 mg%.

## SUMMARY

There is no doubt, from the present series of cases, that the keynote of treatment of these desperately ill Zulu comatose diabetics is :-

(1) Plenty of Intravenous soluble Insulin (were up to 1000 units in 5 hours).
(2) 2 to 3 bottles of plasma given immediately to help restore blood flow to adrenals, brain and kidneys.
(3) 2 to 3 litres of normal saline, and no more to be given quickly into the femoral vein.

SECTION III - 28 (continued)

(4) An indefinite but large amount of 5% Dextrose and water during the next 24 hours, depending upon clinical state, but no more saline.

(5) Potassium citrate and sodium bicarbonate given into the stomach by Ryles tube, rather than intravenously, if the patient cannot swallow.

(6) A bladder catheter, and cover by a broad spectrum antibiotic to combat urinary tract infection.

It is interesting to note that our colleagues in Johannesburg with extensive experience of the treatment of diabetic coma in African people, give 500 units of soluble Insulin intravenously when the patient is first seen, and if there is not material improvement this is followed after one hour with 1000 units. The results in these desperately ill patients are very gratifying indeed.

Needless to say, an energetic search should be made for the cause of the diabetic coma in these cases, but this is generally due to having missed injections for some days.

SECTION III - 29 HYPOGLYCAEMIC ATTACKS.

Insulin resistance and flexibility of dosage is seen to be a much greater extent in the Zulu than would be expected in the European, but not as much as is seen in the Natal Indian. In other words, knife-edge control is not desirable or satisfactory. Consequently hypoglycaemic coma is not a common problem. Many patients on Insulin complain at times (particularly before meals) of very mild hypoglycaemic symptoms, and this is regarded as an urgent sign to lower Insulin dosage. A more severe hypoglycaemic episode in the present series was extreme pugnacity, amounting almost to amok (Case 35). This was a male, aged 45, in whom phases of "amok" were easily controlled by changing the patient from Insulin (40 units of Lente) to Tolbutamide (500 and later 250 mg. three times a day) on which he had subsequently remained clear of glycosuria for 7 years. His symptoms of "amok" were caused by taking his unnecessary Insulin at irregular times in

SECTION III - 29 (continued)

the early mornings, and varying his meal times to fit in with his occupation and exertions as a chemist's delivery boy and factotum. However, remarkable to relate, he became hypoglycaemic on 500 mg of Tolbutamide TDS, but is now well controlled on 250 mg. TDS without glycosuria or hypoglycaemia.

Recurrent mild ketosis is not necessarily a lethal syndrome in the Zulu diabetic, but hypoglycaemia may very well be, as patients' relations are never over speedy about getting them to hospital when they become unconscious, the maxim being "wait and see what happens".

I am entirely satisfied that it is wiser, under our present circumstances, to err on the side of mild reversible bad diabetic control than to approach knife-edge control, as such patients invariably end up in severe hypoglycaemic attacks.

Follow-up Cases.

Case No. 42. was a 32 year old female, who is one of our best patients - tried very hard during the 9 years she has attended, to keep herself under good control. She keeps on having repeated severe hypoglycaemic episodes and is obviously a very labile patient. Attempts to smooth out control with between 80 and 100 units of Lente Insulin, by using the diaguanides, was not successful. However, her health remains remarkably good.

SECTION III - 30 THE DIFFERENTIAL DIAGNOSIS OF GLYCOSURIA IN THE ZULU.

(A) HUNGER DIABETES OR "REFEEDING GLYCOSURIA".

Patients who are severely malnourished eventually develop an intolerance to carbohydrate foods, in that when they do gain access to carbohydrate they develop a glycosuria and hypoglycaemia. In any malnourished population, this type of glycosuria has to be borne in mind. In the first 1250 diabetics of the Clinic, this was seen in only one patient, and this was in a grossly emaciated Indian woman. This is very much a replacement glycosuria and must be borne in mind in any really emaciated patient who shows sugar in the urine.

SECTION III - 30 (continued)

(B) ENCEPHALOPATHIC HYPOTHALAMIC SYNDROMES.

Two such patients were admitted to the King Edward VIII Hospital in the last two years. The syndrome is characterised by coma, hypothermia, hypoglycaemia, glycosuria and acetone in the urine. One of these patients was very nearly despatched by being given a dose of soluble Insulin, in the belief that he was suffering from diabetic coma. The moral of the incident is that never should Insulin be given to a patient who has a rectal temperature of less than 85 degrees F. In each case the condition appeared to be due to excessive imbibition of an extremely powerful intoxicant mixture containing, amongst other constituents, carbide, methyl alcohol and local distillate, drunk after an initial period of "maturation".

(C) CUSHING'S SYNDROME.

Two such cases in children were admitted during 1958 and 1959, and two such cases since then. The correct diagnosis was obvious on clinical grounds. All had glycosuria.

(D) GLYCOGEN STORAGE DISEASE.

One such case was seen in 1959, and presented, with cardiac failure, an enormous liver and a glycosuria. Liver biopsy confirmed the diagnosis.

(E) LIVER DISEASE.

Before glycosuria was considered to be rare in siderosis, but our concepts of diabetes and siderosis have had to be revised in view of the observations of Seftel and his co-workers in Johannesburg, where no less than 20% of their Bantu diabetics have proved to be siderotic. (Pers. comm - 1959).

(F) LAG CURVE AND LOW RENAL THRESHOLD.

In a large subsequent survey of diabetic and non-diabetic Africans in the Republic, these two conditions have been shown to be excessively uncommon. (G.D. Campbell (1963) Confidential Memo for the Life Offices Association Fellowship in Diabetes, No. 4 - available at the Diabetic Clinic, 23, Beatrice Street, Durban.)

## SECTION III - 31 THE PREDIABETIC STATE.

### (A) THE PREDIABETIC STATE IN 68 PAROUS ZULU WOMEN.

The incidence of intrauterine deaths, spontaneous abortions, twins, congenital malformations, neonatal and postnatal deaths is so high in the non-diabetic Zulu women that it is probably not valid to ascribe any of these occurrences to the prediabetic state. Therefore, in this series only the fact of having borne very large babies is regarded as prima facie evidence of this. Few of our patients can remember the weights of their babies at birth, and few of their children were weighed. A series of simple questions was evolved to try and find out whether the child was overweight or not. In the absence of actual birth weights, any child that has not been born with difficulty has been excluded - the Zulu pelvis being "so shocking" as one local Gynaecologist put it, that it would be difficult for any overweight child to be born without difficulty.

Furthermore, the Zulu women have good memories of the size of their children at birth, and by careful questioning, patients were asked if in the belief of the mother, the child was bigger than the newborn children of other women or previous children of their own. In many patients this question was answered in the most graphical manner, indicating that the children concerned were enormously larger than others born by the mother herself or other mothers. Thus I was left with the following information :-

TOTAL NUMBER OF PAROUS WOMEN OUT OF
80 FEMALE ZULU DIABETICS ..... 65

NUMBER OF WOMEN WHO HAD SATISFACTORILY INDICATED THAT SOME OF THEIR CHILDREN HAD BEEN GROSSLY OVERWEIGHT AT BIRTH ..... 49

(Showing that a history of having borne large children is obtained in 75% of 65 Zulu diabetic women.)

SECTION III - 31(continued)

As regards the male diabetics:-

Total Number of Males who remembered the size of their children at birth. ..... 43.

Number of these Male Diabetics, who were confident that children were overweight at birth..... 12.

(B). Boils as a pre-diabetic phenomenon.

12 out of the series of 133 recorded having had boils before developing diabetes.

(C). Carcinoma of the Body of the Uterus.

One Zulu lady, shortly after the closure of the present series, came to the Clinic 2 years after having been treated for carcinoma of the body of the uterus - a very rare syndrome in the Zulu.

(D). Sudden Growth.

In none of the younger patients was there a history of sudden growth in the period immediatly preceding onset of diabetes.

SECTION III - 32 DIABETES AND PARITY.

Here are compared the parity status of (a) 80 female Zulu diabetics in this series:(b)80 age-matched patients at the Gynaecological O.P.D. of the King Edward Hospital:(c)80 age-matched patients at the McCord Zulu Hospl. Gynaecological O.P.D., and (d) 80 age-matched females who were relatives attending patients who had come to the M.O.P.D. of the King Edward Hospl:-

| PARITY | 0 | 1 - 3 | 4 - 6 | 7 - 9 | 10-12 | 13-15 | 16-18 |
|---|---|---|---|---|---|---|---|
| (a). Present series of 80 female Zulu diabetics. | 15 | 20 | 17 | 16 | 7 | 4 | 1 |
| (b). 80 non-diabetics King Edward Gynae OPD. | 22 | 31 | 20 | 5 | 2 | - | - |
| (c). 80 non-diabetics McCord Zulu Hospl. Gynae OPD. | 19 | 34 | 19 | 6 | 2 | - | - |
| (d). 80 non-diabetic relatives attending with MOPD patients - King Edward Hospital. | 20 | 28 | 19 | 11 | 1 | - | 1 |

Note: Women exceeding Para 6 in the Table are as follows:-

(a). Diabetic group - 28. (b). King Edward Gynae Clinic - 7.

(c). McCord Zulu Hospital Gynae Clinic - 8. (d). Relatives of patients attending the King Edward M.O.P.D. - 12. There were less non-parous diabetics.

SECTION III - 32(contd.)

(i). (Figures through the kindness of Dr. G.Batchelor, Durban)
It was in Cape Town, Jackson(W.P.U., (1961). Lancet ii, 1369) first cast doubt upon the possible diabetogenic effect of pregnancy. Previously it had been stated(Pyke, D.A. (1956). Lancet, ii, 818) that the effects of parity alone are enough to account for the true differences in the sex incidence of diabetes - a statement that Jackson set out to investigate, and was unable to substantiate.
The figures from the present series, and age-matched controls are of interest, and a heavy line has been inserted (a) between the non-parous and parous groups, and (b) between the para 6's and those above para 6, and these serve to accentuate the differences between the various groups.

SECTION III - 33 THE ZULU DIABETIC AND OTHER ENDOCRINE DISEASE.

The association of diabetes with other endocrine disorders, particularly thyroid disease, is well known in temperate climates. Endocrine disease other than diabetes, is uncommon in the Zulu people. There are to be seen in the Diabetic Clinic(where medical cases of thyroid disease are seen) a number of patients suffering from goitres which are (a) mostly goitres from iodine-deficiency areas in the coastal mountain ridges running parallel to the sea about 20 miles inland, and (b) conditioned iodine-deficiency goitres such as those associated with puberty, and in whom normal regression has not taken place. In both of these groups, nodular changes always supervene, to an extent far more commonly seen in Europe or America: in 2 such patients of a total of 100, neoplastic changes have occurred. Thyrotoxicosis is extremely rare.

In only one diabetic in this series was there a pathologically enlarged thyroid gland requiring resection which was actually done, and that was case No. 142. This was a very fat young insulin-dependent women(see p. 95) in whom a non-toxic nodular goitre had developed fairly rapidly over 5 years. Exactly one year after resection of the gland for pressure symptoms, she was re-admitted severely ketotic, and she became severely hypolgycaemic during re-stabilisation, with a degree of insulin sensitivity that would hardly have been expected in such a fat diabetic even though she was very young. She subsequently died in irreversable diabetic coma when next admitted. It is of interest to note, that not

SECTION III - 33 (continued)

included in this series was a patient, a Zulu lady, weighing 300 lbs., who, following thyroidectomy, developed mild diabetes.

Amenorrhoea was seen in 16 of 48 menstruating females when they first presented for treatment of diabetes. One patient, as stated above, had had treatment for carcinoma of the body of the uterus, which is a very rare syndrome in the Zulu, before developing diabetes.

Only one case of acromegaly has been reported in a Zulu from the Hospital. As noted in the Section on differential diagnosis, four cases of Cushing's Syndrome have been admitted to the Hospital and treated during the last nine years. In order that there should be information about the numerical relationship between diabetes and other Endocrine disease in patients referred to our Clinic, the following table is appended:-

TABLE SHOWING PROPORTION OF DIABETIC TO OTHER ENDOCRINE CASES SEEN OVER THE FIRST SIXTEEN MONTHS' EXISTENCE OF THE DIABETIC CLINIC OF THE HOSPITAL TO WHOM ALL ENDOCRINE CASES WERE REFERRED.

| | BANTU (Africans) | INDIANS |
|---|---|---|
| DIABETES MELLITUS | 256 (mostly Zulus) | 1100 |
| THYROID DISORDER: (Iodine deficiency, nodular, puberty goitre and carcinoma) | 31 | 28 |
| HYPOTHALAMIC COMAS | 2 | - |
| HYPOPITUITARISM | 1 | -- |
| ADDISON'S DISEASE | - | 1 |
| CUSHING'S SYNDROME | 2 | - |
| LAURENCE-MOON-BIEDL SYNDROME | 2 | - |
| PROGRESIVE LIPODYSTROPHY | - | 1 |
| ACROMEGALY | 1 | 2 |
| HEMIHYPERTROPHY | 1 | - |
| | 296 | 1,132 |

Follow-up Cases:

Case No. 43 a 50 year old obese female, in the 4th year of her attendance and the 8th year of her diabetes, was on good control with Tolbutamide 250 mgms. B.D. having originally been on 500 mgms. T.D.S.

SECTION III - 33 (continued)

This patient had actually been in diabetic coma previously and had been on 90 units of Lente Insulin, but had converted satisfactorily to oral treatment. During the time on Tolbutamide, she developed, quite quickly, a large isthmic goitre but refused to have it operated on, and soon after disappeared from the Clinic, almost certainly due to the fact that we had asked her to submit to an operation.

## SECTION IV. ORGANISATION OF THE DIABETIC CLINIC FOR ZULU PATIENTS - THE DIETARY AND DRUG TREATMENT OF ZULU DIABETICS.

### SECTION IV(A) - 1 GENERAL PROBLEMS UNDERLYING THE TREATMENT OF ILLITERATE AND SEMI-ILLITERATE DIABETICS.

All doctors who see large numbers of diabetic patients will agree that their task, even amongst intelligent and literate people, is not always an easy one. In the better Clinics in the United Kingdom and the United States, there are always a minority of patients that one dreads seeing, generally because they do not take care of themselves, possibly because they have lost all insight into the potential seriousness of their condition. These patients are bad "dieters", bad attenders, and bad about their syringe technique. Coming from the lower economic groups (in comparison with Europeans) of the Zulu community around Durban, the majority of our patients belong to the category of what might be called "bad" diabetics, and those that are in a position to do exactly what is asked of them are in the minority. Many of these "bad" diabetics are honest and genuine patients who try as hard as they can to carry out the diabetologist's instructions, but they cannot diet because they cannot afford high-carbohydrate diets: they cannot avoid injection abscesses because they live in dirty homes and do not wash terribly often, and they cannot always attend the Clinic because of being unable to get away from work or because of transport difficulties.

This, therefore, was earlier background in trying to control the majority of Zulu diabetics, who are underprivileged, and until recently the control of these patients was a nightmare of dietary inflexibility, default and drug indiscretion. This state has been allowed to obtain primarily because doctors have been unable to speak the patient's language, and because of this they have not been in a position to educate the patient precisely about the conduct of the diabetic state. The disinclination of local private doctors to try and treat non-European diabetics has been reflected in the steady flow of patients from hospital and not only from the General Practitioner but from other hospitals alike to the Diabetic Clinic of the King Edward VIII Hospital.

SECTION IV(A) - 1 (continued)

This diabetic problem has been tackled in the following ways :-

(A) That the doctor who sees a Zulu patient should be a reasonably fluent Zulu speaker - and I have seen all these patients personally at every visit, whenever I could, and have tried to memorise as many of their names as I can,

(B) Special propaganda methods were evolved, so the patients, while they are waiting (at their first visit in particular) should be given lectures and demonstrations about dieting, urine testing, and syringe technique. These were given by two Zulu nursing sisters who had been carefully trained in the Clinic.

(C) That all patients who could, should be made to test their urines. This measure is very valuable in impressing upon them that they are suffering from a serious condition, and that they should take care of themselves.

(D) The patient is told that though the condition is a serious one, it can be perfectly stabilised on an out-patient basis - which gives the patient far greater confidence in treating himself.

(E) All patients are told that on whatever treatment they may be, the "pills" or the injections are merely adjuncts to strict dietary regime, and though they may not be able to afford non-carbyhydrate foods, then they must try , to the best of their ability, to stick to the rules we set down for them.

(F) Defaulting is obviated as much as possible by giving each patient, whether he can read or not, a small appointment slip. This has resulted in a follow-up, which is better than can be claimed by many clinics, and this is an example that has now been followed by other specialised clinics.

(G) Finally, as sympathetic an attitude as possible is directed towards the patients.

SECTION IV(A) - 2 THE COLLECTION OF PATIENTS.

The establishment of a Diabetic Clinic at the King Edward VIII Hospital in 1958 resulted in an immediate influx of patients from a number of out-patient departments spread throughout this large Hospital, where many had been attending for some years. In addition, the Clinic was able to glean numbers of patients from the

SECTION IV(A) - 2 (continued)

gynaecological, surgical and especially the ophthalmological wards, but in particular from the medical Out-Patient Department, where gravely overworked doctors, who tried to see 1200 - 1500 patients daily, were only too pleased to refer their diabetics to the Clinic. In this way, 188 Zulu and other Bantu diabetics were collected very quickly indeed (over 2000 in the first 9 years), and it is from this that the present series of 133 was taken.

In an article on diet written for the "Ilanga Lase Natal" (The Zulu Weekly) by my co-author, in the "Handbook to Aid in the Treatment of Zulu Patients" (H.C. and Lugg, 1961, Natal University Press), the following announcement appeared:-

| Zulu. | English. |
|---|---|
| Lapha kwaKhangela kukhonaikliniki elelapha abapethwe ushukela. Abakhona bafuna kakhulu ukuba bonke abaguliswa yisona basondele bahlolwe, balawulwe ngokudla okubafanele ngokungafanele okuzilwayo. Sondelani bakhithi, ningatetemi! (H.C. Lugg, Welfare Officer) | At the King Edward Hospital, there is a clinic where they treat those who have diabetes. The people there are very anxious that all people who are suffering from this condition should gather there to be examined, where they will be told what foods they are permitted to eat, and what not to eat. Come along then - don't hang back! |

This announcement in a widely read paper had the effect of bringing into the Clinic only very limited numbers of patients. Fortunately, or perhaps now in view of the enormous numbers of patients, unfortunately, the Clinic is being widely spoken about amongst rural Zulus and urban Zulus, so much so that many diabetics often make special trips for hundreds of miles to attend the Clinic.

SECTION IV(A) - 3 DIABETIC PROPAGANDA SYSTEMS IN THE CLINIC WAITING ROOM.

Valuable work was achieved by propaganda posters in the Preventive Health Clinics in Durban, by the Institute of Family Health and Community Planning. When the Diabetic Clinic was first started I consulted the Institute as to whether posters would be more effective

SECTION IV(A) - 3 (continued)

than the verbal method of propaganda that I have out lined. In regard to health propaganda posters in semi-literate people, these can be very much of a two-edged sword, and may ingender exactly the opposite effect to that intended. A poster was evolved in 1958, to try and impress upon Zulu people the advantages of obesity. On the one hand, a thin young lady was shown climbing a hill, and soon after, was industriously employed in cleaning her house, and cooking. A very fat lady was depicted struggling and sweating up the slope, and collapsing in a most comfortable looking chair, quite unable to do her house work. Over 90% of the Zulu people interviewed as to their impressions, were certain that the fat lady, being fat, was obviously well-fed, and being well-fed, was obviously rich, and being rich might sweat on going up a steep hill, but when she got to the top, she was at liberty to loll in a comfortable chair, whilst the servant(which she was able to employ, but was not fit to be seen in the picture) did all her housework. In view of the difficulty amongst patients in interpreting such posters, I decided not to use them, and have only displayed pictures of "forbidden"(i.e. high carbohydrate)foods on posters set up in the Clinic waiting Room.

In addition to these methods(and the verbal methods of propaganda), for 5 years I have used a propaganda sheet in an attempt to set out in simple terms for our patients, what we feel are the principal precipitating factors in the emergence of diabetes in the Non-white people in Nat al. I stressed the fact that in rural populations eating only home-ground cereals, that diabetes was extremely uncommon, but appeared in significant proportions in people exposed to urban eating habits, with special respect to refined carbohydrates, such as sugar and white bread of high degrees of refinement. No less than 14,000 of these tracts have been handed out in the Clinic for patients and their relatives, and also in the streets of Durban by the Clinic orderly, especially on days when his work at the Clinic was slack. The per capita consumption of sugar in the Indian people during the 3 years of this campaign actually <u>increased</u> 10%, but there was no increase in the consumption by the Zulus. As the campaign was directed mainly towards the Indians, these results are difficult to interpret, other than to say that the campaign may have resulted in a massive and gratituitous advertisement for the Sugar Industry!

SECTION IV(A) - 4 URINE TESTS IN THE CLINIC, AND TEACHING THE PATIENT HOW TO TEST HIS URINE AT HOME. THE "PROFORMA" STAMP.

URINE TEST RECORD - RUBBER STAMP.

| | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| DATE | | | | | | | | | | | | | | | | | | | |
| TIME | | | | | | | | | | | | | | | | | | | |
| ORANGE BRICK | | | | | | | | | | | | | | | | | | | |
| YELLOW | | | | | | | | | | | | | | | | | | | |
| GREEN | | | | | | | | | | | | | | | | | | | |
| BLUE | | | | | | | | | | | | | | | | | | | |

It was decided from the commencement of the Clinic that all patients, however literate or otherwise, should be taught how to test their urines, and that as many as could be taught should learn how to fill a special testing form. All patients, when waiting in the Clinic ante-room, sat facing a Zulu nurse who tested their urines by means of Benedict's Solution in front of them, and the steps were explained to them. I have not used "Testape" because of cost and paucity of colour, as to the Zulus most shades of green are one. "Clinitest" has also not been used because the tablets go off very quickly in the local high humidity if not carefully kept, and they are also very expensive. Benedict's is of value because of the cheapness, the good range of colour, and because it seems easy to teach even the most illiterate person how to do the test. Strangely enough, the patients prefer the use of a candle to heat the Benedict's urine mixture, and this is an excellent method, especially as we have repeatedly told the patients that it is the proportions of urine-Benedict's that is important, and that if the ratio of 1 : 8 urine to Benedict's is preserved, it doesn't matter how much is used. This results in speed of doing the test and in economy of candle - or spirit lamp.

All patients were given, for a nominal fee:- Spirit lamp and spirits, six tough test tubes and a holder, a dropper. The form for recording results was in the form of a stamp; thus record forms could be made upon any piece of paper that was clean and big enough.

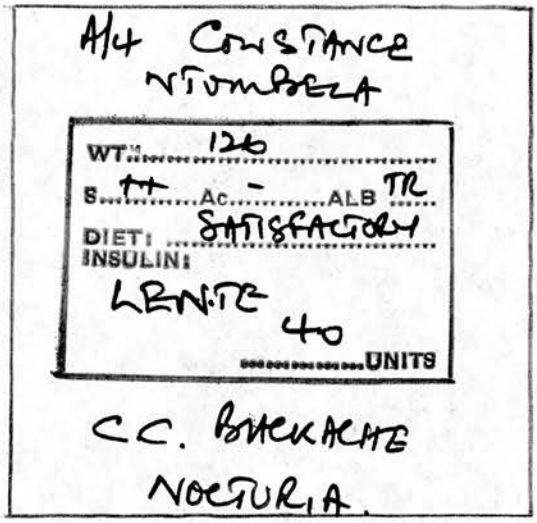
SECTION IV(A) - 4 (continued)

Above is shown the rubber stamp's impression. At the top are spaces for the time and date, and the times that were generally used were morning "a.m." and evening"p.m.", because of difficulty in testing at other times. In many patients, especially in times of initial control and when subsequent control was not satisfactory for various reasons, results of testing on the above chart were of the great help in the assessment of patients controlled as out-patients.

Orange and brick are charted together, as the Zulu name for both is "mbomvu". There is no word for blue in the Zulu language, which is generally referred to as "oluhlaza nenyama" or "green and black", though Zulus are able to distinguish between the two colours. Patients were encouraged to test twice daily, morning and evening, and if they showed brick or orange for any sustained length of time they were told they should return at once to Hospital. Even in the hands of illiterate people, who are tutored by their better educated children, these forms have been of value in assessing progress. In patients who have settled down to reasonable control, I have told them only to test if they are in any way unwell.

In the Clinic a morning random post-prandial urine specimen is collected as soon as the patient arrives, and this is tested for glucose, acetone and for albumin. These results, plus the patient's weight and main symptoms are recorded on a small form made by using another stamp :-

SECTION IV(A) - 4 (continued)




In addition here, the patient's main complaints are also written. This small form is handed to the patient, who presents it, together with his case notes, to the doctor when he gets to the front of the queue.

Here I have given an actual representation of such a record that was presented in the Clinic. Because of the inability of the Laboratory to cope with large numbers of out-patient blood sugar estimations, I have not used them, and only when the Ames Dextrostix became available for finger-prick blood sugars did I make any use of blood sugar estimations, and then only in patients who were obviouslynot well. Any patient with four plus of sugar and three plus of acetone in the urine, would not have a blood sugar done but would at once be admitted to Hospital.

I believe that not only have urine tests been valuable in guiding the patient and me, but that the fact of doing them is in itself a chore that reminds the patient that he must always be looking after himself.

## SECTION IV(A) - 5 THE DOCTOR'S HANDLING OF THE PATIENT.

In the average Zulu literacy is limited, and the lot of the doctor supervising a potentially serious condition in these people is, at times, trying. I feel that the most important factor in the management is that the doctor should be able to speak to them in their own language. Remarkable to say, in this large Hospital, which has no less than 270 full-time and part-time staff, it is probable that not one in thirty can speak to their patients efficiently without the use of an interpreter. I am firmly convinced that medical practice, not only in respect of diabetics, but of all other conditions, could have been greatly improved by insisting that doctors working in the Natal hospitals should speak, at least, a modicum of Zulu.

In the handling of the Zulu patients at the Clinic, I have tried to see them as often as I could, and, in particular, I have tried to memorise their names. Even if they are English speakers, I have still spoken to them in Zulu.

The proper treatment of diabetes imposes hardship on most of our patients, in particular the fat middle-aged patients who find it very hard to restrict carbohydrate and many of whom, because of this, were treated with Insulin over many years. They dislike strict dieting, and on the whole are fond of injections. It is unfortunate that the sulphonylureas have an appetite-stimulating action, and there is evidence that many of our fat diabetics have gained weight on such treatment, unlike the effects of the Biguanides, which have been a great boon in this type of patient. It is true to say that the complaint of great hunger is inevitable if these patients are dieting conscientiously. In all these respects, therefore, very sympathetic handling is necessary, otherwise the patient will default quickly - default being a very good indication of poor handling, or ineffective symptomatic treatment in semi-literate patients.

## SECTION IV(A) - 6 THE CLINICAL EXAMINATION AND CHEST X-RAY.

All patients were thoroughly examined at their first visit and at six-monthly intervals. The results of these examinations are recorded on the following stamp, which is derived from that which was used in the Diabetic Clinic of the Royal Victoria Hospital in Edinburgh :-

A/44 CHRISTOPHER MAPUMULO

| PHYSICAL EXAMINATION | | 6 MONTHLY/~~ANNUAL~~ CHECK | | |
|---|---|---|---|---|
| P. 80 ✓ | HEART: | | R | L |
| BP. 130/75 | (X) ✓ | DP. | ✓ | ✓ |
| Feet. Normal | No m's | PT. | ✓ | ✓ |
| Skin Normal | | | – | – |
| FUNDI. | R | L | | |
| Gr. Type – ve | NAD | NAD | | |
| Cataract Nil | | | | |
| Abd exam. | Other | | R | L |
|  ✓ | ✓ | KJ. | ✓ | ✓ |
| | | AJ. | ✓ | ✓ |
| | | Vib. | – | – |

In the form, I have set out a copy of the routine six-monthly examination of patient A.44 Christopher Mapumulo. In the stamp, "P" = pulse: "BP" blood pressure: "Feet" = the general state of the feet. Under "Heart" there is a space for general comments. (x) represents the position of the apex beat, and there is space for further comments upon the state of the cario-vascular system. "DP" and "PT are the state of the dorsalis pedis and posterior tibial pulses, and there is a space for aberrant pulses - present in 5 - 10% of all people, such as the perforating peroneal which is generally seen represented as "PP". In the space below the stamp, the changes since the last examination can be summarised, or "no change" can be noted. Under "FUNDI" is a very limited space after "Gr" - that is to say "grade" of retinopathy. "Type" would refer in this context to diabetic, hypertensive, or mixed retinopathies. "R" and "L" refer to the right and left fundi, where there is room for fairly detailed

SECTION IV(A) - 6 (continued).

description of observations. "Abd. exam." leaves space for a small pictorial representation of findings there. The title "Other" can be used for unusual findings. "K.J." and "A.J." are knee jerks and ankle jerks, and "Vib." represents the presence or absence of ankle sensation to vibration.

Thus it is easy to summarise at each six-monthly interval changes that have occurred since the last examination.

In addition, all patients had their chest X-rayed at their first visit and six-monthly after. Further, all patients in this series had straight Posterior-Antero X-rays of abdomen to see if calcification of the pancreas was present. This has placed a great strain on a very over-worked Hospital X-ray Department, but in spite of their inundation with work, the Doctors there have been good enough to put up with the large flow of routine X-rays that have come from our Clinic.

SECTION IV(A) - 7 EXPLANATION OF DRUG DOSAGE TO THE PATIENT.

Fortunately most of our patients are on oral therapy, and so instructions about treatment are only those about the taking of tablets, and about the use of saccharine.

Only those patients who will probably require Insulin are admitted to the Wards. For sometime, in numerous lectures to the Nursing Staff, the importance of training diabetics to look after themselves has been particularly stressed. Unfortunately, in this respect momentum has been slow, and patients are frequently referred on discharge to the Clinic after two weeks stabilisation in the Wards, during which time no attempt has been made to teach them injection or urine testing techniques, and this naturally results in a necessity for re-admission for a further two weeks so that this can be done.

It is fortunate that Insulin dosage is remarkably "basic" in the Zulu diabetic, and doses in multiples of 20, 40, 80 or 100 are

SECTION IV(A) - 7 (continued).

found to be eminently satisfactory in the Zulu diabetic. It is interesting to see how many patients are so well controlled on these standard doses without ketosis or hypoglycaemia intervening.

Zulu phrases for explaining drug dosage are set out on p. 157.

## SECTION IV(B) - 1 DIETARY RESTRICTION AND THE DIET SHEET IN ENGLISH. THE "THERAPEUTIC PARADOX" OF OUT-PATIENT TREATMENT.

In the treatment of diabetics coming from the lower economic groups, it is true to say that anywhere in the world dietary restriction is very difficult to institute, and in many instances quite impossible. It is now almost certain that overingestion of refined carbohydrates (the cheapest and most filling foods in advancing countries) is the principal cause for the emergence of diabetes, especially in previously under-privileged people. (Cleave, T.L. and Campbell, G.D. (1966) Diabetes, Coronary Thrombosis and the Saccharine Disease, J. Wright of Bristol. 1st Edition, 14). Thus, as refined carbohydrate is the cause of the disease, it is difficult or almost impossible to eradicate it in trying to alleviate its clinical course. It is true to say that the substitution of saccharine for sugar is the only dietary measure that one can institute in such cases, and it is a sobering thought that were we able to give our patients a high protein diet to the value of the oral anti-diabetic substances that we treat them with, many would be controlled by diet alone.

I devised the following diet "sheet" as being the simplest guide for both races (Zulu and Natal Indians) attending the Clinic. I found that it was best to have it printed in English because patients who could not read, invariably had relations who could, and interpreted the contents of the sheet for them. For sometime I did issue a diet sheet in Zulu, but almost invariably Zulus who could read their own language are able to read English, but for the sake of not having too many "pieces" of paper in the Clinic, I discontinued its use. Patients who were subjected to our verbal anti-diabetic propaganda were told what was written on the sheet, and urged to consult their better educated relations and friends to refresh their memories. The sheet has proved to be one of the most popular regimes in the Hospital, and patients often ask to be allowed to take away extra sheets to give to their friends. Though, at the time of writing, we have in fact 10,500 attending diabetics, we have already given out over 14,000 of these diet sheets.

SECTION IV(B) - 1 (continued)

## THE DIABETIC CLINIC
## KING EDWARD VIII HOSPITAL, CONGELLA
## DIET SHEET.

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**DIABETICS MUST NOT EAT THE FOLLOWING FOODS AT ALL:-**

SUGAR

SWEETS

BREAD (brown or white) except for small amounts.

POTATOES

CAKES.

BEER.

JAM & TINNED FRUITS.

FRIED FOODS.

PUTU, SAMP, MEALIE RICE.

LEMONADE, FANTA, SWEET SYRUPS, or SWEETENED FRUIT JUICES.

**DIABETICS CAN EAT THE FOLLOWING:-**

Three SMALL slices of white or brown bread a day.

All green vegetables and herbs.

Small amouts of RICE (white or brown.

Lettuce and Salads.

Fresh fruit (only ONE banana a day).

Meat, with the fat removed.

Cheese.

Tripes, Chicken, Eggs.

Boiled fish.

Small amounts of broad beans.

BROWN BREAD IS NOT BETTER THAN WHITE FOR DIETING.

BROWN RICE IS NOT BETTER THAN WHITE RICE FOR DIETING.

USE SACCHARINE INSTEAD OF SUGAR - WE WILL GIVE IT TO YOU.

BOIL or GRILL YOUR FOODS RATHER THAN FRYING THEM.

EAT THREE EQUAL SIZED MEALS A DAY.

DRINK TWO GLASSES OF WATER BEFORE EACH MEAL.

USE VINEGAR INSTEAD OF SALAD DRESSING.

IF YOU ARE HUNGRY TAKE TEA OR COFFEE WITHOUT SUGAR, OR GREEN LEAF VEGETABLES.

THE TWO IMPORTANT RULES OF THE DIABETIC ARE :-

DIET CAREFULLY - KEEP YOUR WEIGHT DOWN.

ATTEND THE CLINIC REGULARLY FOR SUPERVISION OF YOUR ~~SUPERVISION OF YOUR~~ TREATMENT.

THE CLINIC IS ON WEDNESDAY MORNINGS AT 9 O'CLOCK.

ALWAYS BRING YOUR SYRINGE AND EMPTY INSULIN BOTTLES WITH YOU.

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SECTION IV(B) - 1 (continued).

This diet sheet has, in many patients been satisfactory. Though it is often said that attempts to diet fat middle-aged Zulu patients, who subsist mainly on carbohydrates, are bound to fail, the accent has been laid on the necessity for never eating sugar, and as many of our Zulu diabetics have a very sweet tooth they are always given a liberal supply of saccharine in the hope that they will use this liberally in tea and cooking, although its use in cooking leaves a bitter taste. On one or two patients weight losses of up to 30 lbs have been achieved at the same time as we have been stabilising them, specially where Biguanides have been used. Most of our patients, fat or thin, tend to gain weight on sulphonylurea therapy.

Anorectic agents are generally very expensive, and with the object of trying to cut drug expenses in the Clinic as much as possible, I have always tried to avoid using them. However, in 1961 I was given a large supply of a drug for clin cal trial (Ciba - "Ba - 28,616") which we found to be effective when compared with patients used as controls, and as a measure of their effectiveness was the great demand in patients who had been treated with this drug, and the outcry in the Clinic when our supplies ran out! Once again, money spent on these very expensive substances if used in the purchase of non-carbohydrate foods, might help greatly in enforcing satisfactory dietary restriction.

## SECTION IV(B) - 2 INSULIN THERAPY AND SYRINGE TECHNIQUE.

Before the wholesale swing of patients away from Insulin to oral agents, injection abscesses were troublesome, not so much by virtue of their frequency but because of the fact that they lead to severe ketosis in a very short time. The provident finding of insulin-independence in many of our patients was welcome as it meant that it would be possible to convert patients away from their insulin.

However, I found that with a regular Clinic Staff the standards of syringe and techniques improved greatly in those still compelled to take insulin, and it is plain that even in illiterate people, short tutorials by a trained Zulu Staff Nurse at each visit, helped with both syringe hygiene and with general cleanliness of the patients. Because of the danger of not washing out syringes properly when they were stored in spirit, I encouraged patients to keep their syringes in a clean cloth and simply rinse them out with warm water before and after use. Patients were encouraged to boil their syringes weekly. These measures seemed to be perfectly adequate. Needles seemed to blunt more quickly than in European clinics and patients were always given a liberal supply of these. Unfortunately proper insulin syringes were not always available and we often had to teach patients to use unsuitable syringes. With a minimum of trouble this was satisfactory. Ether was given to patients and plenty of cotton wool for swabbing purposes. Because of the remarkable uncommonness of local lipoatrophy, it seems unnecessary to teach the use of more than two or four injection sites - arms and legs being the most favoured.

## SECTION IV(B) - 3 USE OF THE ORAL HYPOGLYCAEMIC AGENTS - EFFECTIVENESS.

I have reported very fully on the use of the oral anti-diabetic agents in non-white diabetics in Durban (Campbell, G.D. (1963). S.Afr.Med.J., 37, 48, 1204), and specifically upon their toxicity in the same people (Campbell, G.D. (1961). Proc. 4th Intern. Diab. Cong. (Geneva), 776). Further observations on large numbers of patients are included in an account of the oral treatment of diabetes in the Tropics, now in preparation (Campbell, G.D. (1968).

SECTION IV(B) - 3 (continued).

The Oral Hypoglycaemic Agents - Pharmacology and Therapeutics, The Academic Press - London and New York. Chapter 7).

This table shows the methods of treatment at the three main stages of this study (1) When patients were first seen - up till 5th Feb. 1959, (2) at the end of a minimal follow-up of one year: and (3) at the end of the whole follow-up 5th February, 1968. All methods of treatment that have ever been used by me on the present series are inserted in the table, though at the specific times mentioned, some of these regimes were not, in fact, in use.

(SEE TABLE)

SECTION IV(B) - 3 (continued)

| | PATIENTS ON ORAL TREATMENT | TOLBUTAMIDE | | | | | | CHLORPROPAMIDE | | | A/H | | DBI. | | GP | | COMBINED TREATMENT | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 2 tds | 1½ tds | 1 tds | ½ tds | ½ bd | ½ dly. | 1 bd. | ½ bd. | ½ dly. | 1 tds | ½ tds | 50 mg.bd. | 50 mg.dly. | 1 tds. | ½ tds. | PBI & Tolb. | PBI & Chlor. | Other. |
| WHEN FIRST SEEN | 45 out of 133 till 5.2.1959 | 6 | 10 | 28 | 1 | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| AFTER ONE YEAR | 89 out of 133 at 5.2.1960. That is, at the end of the qualifying 1 year attendance | - | - | 49 | 33 | 6 | 1 | - | - | - | - | - | - | - | - | - | - | - | - |
| WHEN LAST SEEN | 100 out of 133 as of 5.2.1968, or at last visit to the Clinic. | - | - | 24 | 16 | 17 | ± | - | 13 | - | - | - | 12 | 18 | - | - | - | - | - |

Reference to Table: "A/H = Acetohexamide
"DBI"= Phenformin
"G/P"= Metformin.

"Combined Treatment" = PBI + Tolbutamide
or PBI + Chlorpropamide.

SECTION IV(B) - 3 (continued.

NOTE:-

(1) In 1958 and 1959 Tolbutamide was the only drug that we had available for the oral treatment of diabetes in the Clinic, and it can be seen that large dosage was the custom at that time.
(2) Chlorpropamide only became freely available in our Hospital Dispensary after the beginning of 1960, and was thus not used at the commencement of the study after the first year's follow-up.
(3) Though not noted, there were other regimes used in this series. During the course of this study four patients were on Acetohexamide 250 mgms. (½ tablet TDS), all being started on 500 mgms TDS, but unfortunately supplies were not continuously available at the Hospital. Five were on Metformin, in two instances the drug being used to attempt better insulin control. In the Zulu this does not appear to be a very effective drug, especially when compared with Phenformin. Fifteen were on combined therapy with Phenformin and Tolbutamide (9), and Chlorpropamide (6), but I did not consider that the expense of combined therapy merited further use of combined therapy, especially where dietary failure was the main cause of poor control. (4) The change from large to small Tolbutamide dosage over the course of the study was striking. (5) Chlorpropamide proved useful in thirteen patients, and was only used in cases of thinner middle-aged people if Tolbutamide control did not appear satisfactory because (a) of possible hepatoxic effects in a group of people in whom liver disease might be believed to be unduly common, and (b) because I try never to use the sulphonylureas in insulin-independent patients who are overweight, because this causes increases in weight. (6) Phenformin was used in the timed-disintegrating capsule form (DBI-TD) in thirty cases (in twelve 2 capsules and in eighteen 1 capsule daily). This is an almost ideal anti-diabetic substance in patients who cannot or will not diet.
(7) Toxic effects in the Zulu, as compared with the Indian and European people from the oral anti-diabetic agents is negligible.
Criteria of control are set out on p.122 and results on p. 123.

## SECTION IV(B) - 4 CRITERIA OF CONTROL.

Elsewhere (Campbell G.D. (in proof) Chapter 7 - Oral Treatment of Diabetics in Tropical Countries - Oral Hypoglycaemic Agents - Pharmacology and Therapeutics, Academic Press, London and N.Y.C.) I have set out criteria for satisfactory control of tropical diabetics in both the well-appointed and the poorly-appointed Clinic. The poorly appointed Clinic I regard as one where large numbers of patients have to be seen by small numbers of doctors, and the facilities and time for taking off blood specimens for blood sugar levels is not available. Under such circumstances I take into account the following features :-

(1) Have the patient's symptoms improved?
(2) Is there less sugar in the urine than before treatment?
(3) Has the patient noted any side-effects?
(4) Is the patient satisfied with the treatment?

I regard (4) as being the most important as it virtually summarises (1), (2) and (3) and is the chief factor in obtaining a good follow-up. As a clinical parallel to these questions, I recommend the following as guides to assessing response :-

EXCELLENT: completely asymptomatic: always aglycosuric whilst on treatment.

GOOD: completely asymptomatic: occasional, even if heavy, glycosuria.

FAIR: not completely asymptomatic, but greatly improved: post-prandial glycosuria invariable: weight still higher than when treatment was begun.

POOR: symptoms not relieved at all: heavy glycosuria constantly present: often further weight is lost, especially if ketones are present in the urine.

Though anyone who assesses his responses on clinical grounds alone will be criticised for "lack of biochemical control", it is remarkable how well clinical results correlate with those gauged upon laboratory investigations. In defence of standards of supervision and control that might not be considered entirely satisfactory in Clinics in Europe and America, I would like to quote Prof. Tulloch, generally acknowledged as the principal expert on tropical diabetics,

SECTION IV(B) - 4 (continued)

when speaking of my work in Durban he says "Admittedly, his criteria of response are less rigid than would be accepted by the Joslin Clinic, but in the circumstances in which he finds himself, they are realistic and practical". (Tulloch, J.A. (1962). Diabetes Mellitus in the Tropics. E and S Livingstone, Edinburgh and London, 191).

SECTION IV(B) - 5 METHODS AND RESULTS OF TREATMENT.

1959 saw the beginning of the massive swing from Insulin to the oral agents in our Clinic. In the following table, therefore, I have summarised the method of treatment in the 133 Zulu diabetic patients at the end of the first year's follow-up.

TABLE SHOWING METHODS OF TREATMENT IN THE VARIOUS DIABETIC GROUPS AT THE END OF THE FIRST YEAR'S FOLLOW-UP.

| DIABETIC | SEXES | DIET ALONE | INSULIN (Units) 0 - 20 | 20 - 40 | 40 plus | TOLBUTAMIDE ½ or 1 TDS |
|---|---|---|---|---|---|---|
| YOUNG INSULIN DEPENDENT | MALES (16) | - | - | 6 | 10 | - |
| | FEMALES (16) | - | - | 6 | 8 | 2 (xx) |
| MIDDLE-AGED. | MALES (32) | 1 | - | - | 4(x) | 27 |
| | FEMALES (56) | - | - | 2(x) | 2(x) | 52 |
| SENILE | MALES (5) | - | 1 | - | - | 4 |
| | FEMALES (8) | 2 | 1 | - | 1 | 4 |
| TOTALS | 133 | 3 | 2 | 14 | 25 | 89 |

(xx) The two original "Insulin-independent" young diabetics of the "Tropics".

(x) Patients who should not ordinarily have been on insulin but were on it for various reasons (complications).

SECTION IV(B) - 5 (continued)

This shows that by this time 89 out of the 133 were on oral therapy, 3 on diet alone, and 41 were on insulin. This compared with 45 on oral therapy, none on diet alone and no less than 88 on insulin when patients were first seen. Since the first year's follow-up further patients have been given trials on oral therapy with success, with what I have regarded as being reasonable diabetic control. The following table summarises the overall changes in the whole series in methods used in attempts to control the diabetes, (a) when the patient was first seen, (b) at the end of a minimum one year's follow-up, and (c) when the patient last attended, when he or she died, or disappeared.

| | When first seen (8/8/58 - 15/2/59 | At end of 1 yr. 15/2/60 | At end of the follow-up. 15/2/68. |
|---|---|---|---|
| Oral Therapy (Single or combined) | 45 | 89 | 100 |
| Insulin | 88 | 41 | 33 |
| Diet alone | - | 3 | - |

The table emphasises the fact that there was a large swing from insulin to oral therapy in the first year of follow-up.

TABLE SUMMARISING METHODS OF TREATMENT BY DIABETIC TYPE AT LAST VISIT.

| DIABETICS | | DIET ALONE | INSULIN 0-40 | INSULIN 40 Plus | Tolb. ½ BD | Tolb. ½ TDS | Tolb. 1 TDS. | Phen-formin | Met-formin. | Chlorp rope. |
|---|---|---|---|---|---|---|---|---|---|---|
| Young mainly insulin dependent | MALE | - | 7 | 9 | - | - | - | - | - | - |
| | FEMALE | - | 6 | 8 | - | - | 2 | - | - | - |
| Middle-aged. | MALE | - | - | - | 3 | 4 | 9 | 12 | - | 4 |
| | FEMALE | - | 1 (Case No.7) | - | 14 | 7 | 8 | 18 | - | 8 |
| Senile | MALE | - | - | - | - | - | 5 | - | - | - |
| | FEMALE | - | - | 2 | - | 5 | - | - | - | 1 |
| | | DIET | INSULIN | | THE VARIOUS ORAL REGIMES | | | | | |
| | | - | 33 | | 100 | | | | | |

Note: (a) No patients on diet alone.
(b) Smaller doses of Tolbutamide.
(c) Increased use of Phenformin.

SECTION IV(B) - 5 (continued)

STANDARDS OF CONTROL BY DIABETIC GROUP AND SEX
(a) at end of first year (b) when patient was last seen.
(Excellent, good, fair, or poor. p. 122)

| | | After first year follow-up | | | | When patient last seen. | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | EXCT. | GOOD | FAIR | POOR | EXCT. | GOOD | FAIR | POOR. |
| YOUNG | MALE | - | 9 | 5 | 2 | - | 9 | 4 | 3 |
| | FEMALE | - | 8 | 7 | 1 | - | 9 | 5 | 2 |
| MIDDLE-AGED | MALE | $3_{(x)}$ | 14 | 10 | 5 | 5 | 13 | 8 | 6 |
| | FEMALE | $8_{(x)}$ | 20 | 26 | 2 | 7 | 21 | 23 | 5 |
| SENILE | MALE | - | - | 5 | - | - | - | 5 | - |
| | FEMALE | - | 2 | 4 | 2 | - | 2 | 4 | 2 |
| TOTALS | | 11 | 53 | 57 | 12 | 12 | 54 | 49 | 18 |

(x) = all very mild diabetics.

There is negligible difference between control at the end of one year and control when the patient was last seen: thus control seems to have been established - not to change subsequently - at the end of the first year.

SECTION IV(B) - 6 SURGERY IN THE ZULU DIABETIC.

By far the majority of patients referred for surgery were patients who needed cataract operations, and these were discussed fully on p. 87. Two pregnant diabetics were delivered by Caesarean section (p. 129). The following table shows the number of operations that were done under general anaesthetics on patients in this series :-

| | | |
|---|---|---|
| Cataracts | Senile | 26(see p.87). |
| | "True" | 12(see p. 89). |
| Pregnancy | Caesareans | 2(see p.129). |
| Orthopaedic | Broken leg | 1( 4 Ops.). |
| Breast abscesses | | 5(see p.80). |
| Drainage of Massive Abscess | | 1(see p.80). |
| Drainage of Palmar Space Infection | | 1(see p. 81). |
| Thyroidectomy | | 1(see p. 101). |
| | TOTAL | 49 |

Case No. 105 was a 58-year old male who was knocked down by a motor car, and who had a series of operations (4) for treatment of comminuted fractures of his right femur. During the time in Hospital he was changed from Tolbutamide to Insulin, and both the operations and his convalescence covered by this drug. After discharge, he was put back on to 250 mgms. of Tolbutamide daily with good diabetic control.

Case No. 142 was a very obese female who developed a huge gluteal abscess from an infected injection of insulin. This was incised and drained under general anaesthetic, the operation being covered with soluble insulin. She recovered well from this, but later died in Hospital from Diabetic coma.

Other procedures :-

Case No. 133, was a 78-year old male with a huge hydrocoele, which was operated upon under local anaesthetic block, and removed. The patient did very well, and did not need to be changed from Tolbutamide to Insulin for the procedure.

Preparation of Patients for Surgery Under General Anaesthetic:

(Taken from Textbook in Preparation;- "Clinical Medicine in Africans in Southern Africa, E. and S. Livingstone. Ed. G.D. Campbell. Chapter on "Diabetes and Thyroid Disease").

SECTION IV(B) - 6(continued)

The most important thing in preparing the Zulu Diabetic for Surgery is to try and impress upon surgical colleagues that all diabetics should be the first patients operated on every day. However, with gravely overcrowded theratres this is not always feasible.

In helping the Surgeon, Anaesthetist and Obstetrician there is no better aid than the "Dextrostix"(Ames) for rapid blood sugar estimation from finger prick blood.

(a) Insulin-dependent Patients.

If the operation is an elective one, then the patient should be admitted to Hospital 2 days before it is to be done, and the depot insulin replaced by twice or thrice-daily soluble insulin. As a rough guide, in Zulu patients on 40 units of Lente insulin daily, the patient will probably be well controlled on 16 - 20 soluble in the morning, and 16 in the evening.

On the morning of the operation, it is wise to ask the anaesthetist to put up a drip of glucose-saline, and to give the patient 2/3 of his yesterday's A.M. dose of soluble insulin 45 minutes before the operation. This will almost certainly result in blood sugar levels in the operation that are within "reasonable" limits. At the end of the procedure, finger-prick blood should be tested as the patient leaves the theatre. As a general rule, if the patient is eating satisfactorily, depot insulin should be started the next day. I am thoroughly satisfied with Zulu diabetics, that there is no place for the exquisite "knife-edge" control with insulin, as paradoxically, periods in the Wards on twice daily insulin often give poorer control than that seen with the single daily dose of Lente Insulin.

(b) Insulin-independent patients.

Where control is reasonably good, the operation should proceed as if the patient is a non-diabetic, though it is wise to have a saline drip in place. With the cataract cases, it is wise to keep post-operative blood sugar levels as low as possible for at least one week, even to the extent of keeping the patient mildly hypoglycaemic with the single daily dose of depot insulin, unless one is entirely satisfied by taking random blood sugars, that post-prandial levels are quite satisfactory on oral therapy. Further, it must be remembered that these insulin-independent diabetics are extremely resistant to insulin, and if oral control is not satisfactory, then doses often exceeding 60 units of Lente Insulin daily are necessary, and the

SECTION IV(B) - 6(continued)

absence of hypoglycaemia in such cases is striking. Ideally, these insulin-independent patients should be admitted two days before the operation, to ensure that random blood sugar levels on oral therapy are satisfactory: if they are not, then insulin must be used without hesitation - soluble insulin for the day of the operation, and a return to lente insulin the next day. I cannot help believing the contention that the prime cause of poor results in cataract surgery in the Zulu people is poor control of the diabetes - and such failures in patients in poor control are too numerous for complacency. Further it is wise to be extra-vigilant in stopping visiting relations' bringing in mineral waters and biscuits, which are, unfortunately, only too readily available at the "Tuck Shop" of most large African Hospitals.

SECTION IV(B) - 7 PREGNANCY IN THE ZULU DIABETIC.

In this series, I have been able only to supervise two pregnant patients (Cases No. 4 and 14). Though another patient was delivered of a live baby when on out-patient treatment on insulin, this delivery took place at home and was per vias naturales.(Case No. 170).

The two cases(Nos. 4 and 14) are of considerable interest, as they were the first cases in which we first found that diabetes in pregnancy could be controlled by oral therapy - and here I refer to pregnancy in the established diabetic, and not to glycosuria of pregnancy. Indeed these two cases were the first that we described under the name "the insulin-independent young diabetic of the tropics".(Campbell, G.D., and McNeill, W.G.(1959). Brit. med. J., ii, 634).

Case No. 4 was a 19-year old Zulu girl who had been diabetic for 3 years and who had been treated fairly satisfactorily on a single daily dose of Lente insulin(40 units) for all that time. Unmarried, she unfortunately became pregnant, and became very lackadiasical about her diabetes control, in that for some unaccountable reason, she would go for up to 2 weeks without giving herself any insulin. The effect of this was to cause a very heavy glycosuria, but ketones never appeared in her urine. Finally at about the 26th week of pregnancy, she was put on to Tolbutamide 500 mgms TDS, with remarkable improvement in her control, and on this dosage she was taken to the 36th week, when she was delivered of a healthy infant by Caesarean section. Subsequently, she was well controlled on the same dose of Tolbutamide, and in the

SECTION IV(B) - 7(continued)

many years that we have been following her, has not become pregnant again. However, there have been short times when she has had for some reason or other - such as bad attacks of flu - to be put on to Insulin again, but she is much more commonly off insulin than on it. Periods of needing are never heralded by the appearance of ketones in her urine and it is generally on the grounds of heavy glycosuria, loss of weight, and feeling umwell, that she is changed to insulin from oral therapy. Glucose tolerance tests during a short period of diagnostic cessation of treatment left no doubt as to the correct diagnosis of diabetes.

Case No. 14 was a thin 29-year old lady, who had had diabetes for 4 years and had been reasonably well controlled on a single daily dose of lente Insulin during that time. Two previous pregnancies under Insulin control had ended in stillbirths. At the end of the 20th week of pregnancy, it appeared obvious that she was not looking after herself properly, in that she was not taking her Insulin for periods of up to two weeks, but she refused admission to Hospital. Again as in Case 4, in desperation she was put on to Tolbutamide 500 mgms. TDS, and to my enormous surprise came back a week later in near-perfect control of her diabetes: her out-patient urine testing chart showed that she was totally free of urine sugar, and she said that she was "feeling very much better". She was delivered of a live baby by Caesarean section at the 37th week, and subsequent to this has been very well controlled on the same dose of Tolbutamide.

Case No. 170 disappeared for a year, and returned to the Clinic with the news that she had been delivered of a live child in a country Hospital whilst still on Insulin.

A 45-year old Zulu lady(not in this series), had a succession of babies whilst on oral therapy( confirmed by her employer - a Zululand G.P.) so much so that when I was last in that area, I was told that she had just had her 12th. live baby whilst on oral therapy, without any miscarriages or still-births, and he had indeed to terminate her employment, as she "was always pregnant". All these deliveries were while she was on Tolbutamide 500 mgms TDS, and all were per vias naturales. The problem of young Tropical and Sub-tropical Diabetic women who do not require insulin during pregnancy has been fully summarised. (Jackson, W.P.U., and Campbell, G.D., Notelowitz, M., and Blumsohn,L. 1962. Diabetes, Vol. 11,(Suppl.), pp. 98 - 101).

SECTION IV(B) - 8 DIABETES IN ZULU CHILDREN.

There were no children in the present series of diabetics. Subsequent to the collection of this series I have had the opportunity of treating one such case. It is true to say that in the non-literate and lower class Zulu family, it is quite impossible to keep these patients alive for more than a few months. One such case spent no less than 10 months consecutively in Hospital, and was re-admitted two weeks after discharge in irreversible hypoglycaemic failure, from which she eventually died some days later. Insulin-independence such as I have encountered in the rather older (19 year Zulu diabetic - p. 129) and in even very young Natal Indians (lowest age - Case No. BSI/2407 - M. Sidiq), being $1\frac{1}{2}$ years, I have not recorded in the young Zulu. Thus it is probable that diabetes in young Zulus, being more an Insulin-dependant state than in Indians, almost certainly being not brought to Hospital in time for treatment, would be invariably fatal.

In a visit to the Baragwanath Hospital Diabetic Clinic, I have seen diabetic children from upper class homes being well and satisfactorily treated. It is obvious that home background is of paramount importance here.

SECTION IV(B) - 9 STANDARD OF FOLLOW-UP.

If, as I believe in the Zulu people, that the patient's being satisfied with treatment is one of the most important criteria in assessing control, then it is probably fair to say that (a) regularity and (b) to a lesser extent, length of attendance will be a fair indication of whether we are helping our patients or not - short of those who have defaulted or died. Though only one-quarter of the series were still attending at the end of the nine years, a breakdown of regularity of follow-up by sex and diabetic group is of interest :-

SECTION IV(B) - 9 (continued)

REGULARITY OF ATTENDANCE

(in attempted 9-year follow-up)

(Regardless of length of Attendance).

| | | VERY POOR (More than 1 year between successive visits | MODERATE (Never more than 1 year, but over 6 months between visits | GOOD (Never more than 6 months between visits |
|---|---|---|---|---|
| Young Insulin dependent | Male (16) | - | 5 | 11 |
| | Female (16) | 3 | 1 | 12 |
| Middle-aged | Male (32) | 3 | 9 | 20 |
| | Female (56) | 5 | 17 | 34 |
| Senile | Male (5) | - | 3 | 2 |
| | Female (8) | 1 | 5 | 2 |
| | TOTALS (133) | 12 (9.0%) | 40 (30.1%) | 81 (61.0%) |

The standards of attendance recorded are really remarkably high, and testify to the fact that even if total duration of attendance might not be considered satisfactory (31 out of 133 patients attending at the end of 9 years), when patient did attend, they did so with commendable regularity; thus I believe that we succeeded in this instance and under our circumstances, in impressing an insight of the disease into these patients.

I have set out details of default by sex and diabetic group of the whole series over the nine-year follow-up:-

SECTION IV(B) - 9 (continued)

| ATTENDANCE STUDIES (9 YEARS) | | END OF CONSECUTIVE YEARS. | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | 1 | -2 | -3 | -4 | -5 | -6 | -7 | -8 | -9 |
| YOUNG INSULIN DEPENDENTS | Attending at end of consecutive years | 32 | 20 | 19 | 16 | 13 | 11 | 10 | 10 | 10 |
| | Known as dead | - | 3 | - | - | - | - | - | - | - |
| | Unknown | - | 9 | 1 | 3 | 3 | 2 | 1 | - | - |
| MIDDLE-AGED | Attending at end of consecutive years | 88 | 59 | 48 | 37 | 32 | 27 | 22 | 21 | 20 |
| | Known as dead | - | 1 | - | - | - | - | - | - | 1 |
| | Unknown | - | 28 | 11 | 11 | 5 | 5 | 5 | 1 | - |
| SENILE | Attending at the end of consecutive years. | 13 | 5 | 2 | 2 | 2 | 1 | 1 | 1 | 1 |
| | Known as dead | - | 1 | - | - | - | - | - | - | - |
| | Unknown | - | 7 | 3 | - | - | - | - | - | - |
| | TOTAL ATTENDING AT END OF CONSECUTIVE YEARS. | 1st | 2nd | 3rd | 4th | 5th | 6th | 7th | 8th | 9th |
| | | 133 (a) | 84 | 69 | 55 | 47 | 39 | 33 | 32 | 31 |
| | Percentage attending at end of consecutive years. | 100 | 63.3 | 51.9 | 41.4 | 35.4 | 29.4 | 24.8 | 24.1 | 23.4 |

(All percentages to 1 significant figure)

(a) All patients followed for at least 1 year.

SECTION IV(B) - 9 (continued) STUDIES OF DEFAULT BY CONSECUTIVE YEARS

In contrast are shown annual decreases in attendance by sex and diabetic group over the full follow-up :-

| | | ENDS OF CONSECUTIVE YEARS | | | | | | | | | At end of 9 years. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 1 | -2 | -3 | -4 | -5 | -6 | -7 | -8 | -9 | Attending. | Dead | Unknown. |
| INSULIN-DEPENDENT | MALE | 16 | 9 | 1 | - | 2 | 1 | - | - | - | 3 | 2 | 11 |
| YOUNG | FEMALE | 16 | 3 | - | 3 | 1 | 1 | 1 | - | - | 7 | 1 | 8 |
| MIDDLE-AGED | MALE | 32 | 9 | 6 | 3 | 3 | 2 | - | 1 | - | 8 | 1 | 23 |
| | FEMALE | 56 | 20 | 5 | 8 | 2 | 3 | 5 | - | 1 | 12 | 1 | 43 |
| SENILE | MALE | 5 | 4 | 1 | - | - | - | - | - | - | - | - | 5 |
| | FEMALE | 8 | 4 | 2 | - | - | 1 | - | - | - | 1 | 1 | 6 |
| | | | | | | ANNUAL DECREASES | | | | | | | |
| TOTAL | | 133 | 49 | 15 | 14 | 8 | 8 | 6 | 1 | 1 | 31 | 6 | 96 |
| Annual decrease (percentages) in series | | 100 | 36.8 | 11.3 | 10.5 | 6.0 | 6.0 | 4.5 | 0.8 | 0.8 | 23.4 | 4.5 | 72.2 |

(All percentages to 1 significant figure).

NOTE: All patients followed for at least 1 year.

Over 1/3 of the patients disappeared in the second year of the follow-up proper - that is to say, the year after the full year follow-up that qualified patients for inclusion in the series.

SECTION IV(B) - 9 (continued)

It is difficult to know how the default rate would compare with a diabetic clinic in the more advanced countries, but the attendances of the Zulu patients in this series have not been as good as those that we have experienced with Indian patients at our Clinic. Reasons for this poor follow-up over nine years are suggested :-

(1) The earlier fee of 40 cents (4/-) and more lately a much higher fee to attend, with the exception of the truly indigent patients.

(2) Crowding of the Clinic due to large numbers of patients and shortage of doctors.

(3) Lack of insight in some patients about the seriousness of their disease.

(4) Migration back to the rural areas, or movements associated with changes in occupation, or marriage.

(5) Drift to other hospitals and clinics which are not so crowded.

(6) Transport expense and difficulties.

(7) Non-co-operation of certain employers, who withhold pay for days spent at the Clinic.

(8) Death, which is not reported to the Clinic. This is the probable explanation of brittle-insulin dependent patients who migrate back to the rural areas. A number of cases disappeared shortly after heavy albuminuria was noted. Was this a measure of sudden vascular degeneration?

(9) Inability to get further insulin supplies. In three cases the ominous entry at the last visit was "ran out of insulin and couldn't get back to the Clinic". This almost certainly happened again.

(10) Lack of rapport with other hospitals and clinics due to staff shortages.

(11) Some patients definitely defaulted on being given oral treatment, rather than being left on insulin.

Follow-up Cases: (showing social reasons for disappearance from the Clinic).

As an example of reaction towards blood tests, I would quote Case 120, who was a 48-year old female, who, in the sixth year of her attendance, failed a therapeutic trial of Tolbutamide (500 mg TDS)

SECTION IV(B) - 9 (continued)

in an attempt to wean her off 30 units of Lente Insulin. She was put back on to Insulin again with good control, and came in for studies to estimate her plasma iron levels. She objected greatly to this and did not appear again in the Clinic.

Case No. 137. A 64-year old female, was overweight and was on 60 units of Lente Insulin and was converted with profit to Tolbutamide 500 mgm TDS. At her last visit she said it was her intention to go off for her holiday in the country, and she had never reported since.

Case No. 46. A 46-year old female, in the second year of her attendance and the fourth of her diabetes, was a poor patient in that she would run out of Insulin and not worry to come back to the Clinic. She was admitted at her last visit very ill indeed in severe ketosis, but not coma. Following discharge from hospital she was admonished about looking after herself, but was never seen again.

Case No. 116. A female aged 70 yrs, was subjected to liver biopsy which showed a mild post-necrotic scarring. Following this, I believe, because she did not like having a liver biopsy done, she went home and never appeared in the Clinic again.

Case No. 3. A 43-year old male, was in poor control on Insulin. A trial of oral therapy failed, and he had to be put back to Insulin. At his last visit, after attending two years, control was very poor; he refused admission to hospital and never appeared in the Clinic again.

Case No. 99. A 34-year old male, disappeared after a period of extremely poor control as an out-patient on Lente Insulin, having refused to be admitted to hospital.

Case No. 145. A 34-year old male, was well controlled on Insulin but disappeared after going home to help some relations plough their farm , possibly due to the fact that he was unable to obtain Insulin after some months in a remote area.

Case No. 177. A rather stupid 39 year old male, who was obviously dependent on Insulin, refused to inject himself any more, demanded oral treatment, which was given to him with reluctance, and he was never again seen in the Clinic.

SECTION IV(B) - 9 (continued)

Case No. 174. A 41-year old female, who attended for 1½ years, suddenly went into very poor out-patient control with 50 to 60 units of Lente Insulin. She refused hospital admission, and was never seen again.

---

By the end of this study, it was plain that attempts to treat patients by diet alone had been abandoned, chiefly, as one patient put it, that he "could buy saccharine more cheaply than the fee charged at the Clinic". Thus all patients were given some form of drug treatment, though it was only in few that this was not necessary. It is not possible to say if patients were better off in the long run after the wholesale swing to oral therapy from insulin: on the one hand, use of insulin was not entirely safe in the hands of certain of my patients, whereas on the other hand, oral therapy cannot be said to be suitable for patients who will not or cannot diet.

I have been greatly taken aback by the tide of tuberculotics that has appeared through the course of the 9 years follow-up: whereas 6 of the original 133 patients had had tuberculosis at the beginning of the follow-up, no less than 8 further cases were known to have developed the disease in subsequent study - thus, excluding those who disappeared who had developed tuberculosis, no less than 14 out of the 133 had significant tuberculosis requiring therapy in Hospital. Was this perhaps a measure of the sustained higher blood sugars as compared with the occasional normal levels that are found in the patient on insulin? One thing is certain in the Zulu diabetic and that is that cases of tuberculosis do better if changed from oral to insulin therapy in Hospital. Again, there is little evidence to show conclusively that the "true"(Kimmelstiel-Wilson) nephropathy is due to bad control. Whereas, this condition was very rare when the study started, there seems to have occurred a generous crop(real or presumed) during long-term follow-up. I believe firmly, that satisfactory dietary control, if properly imposed, would result in a dramatic fall in patients attending the Clinic.

SECTION IV(B) - 9 (continued)

Contrary to practice in more socially advanced people, the most important therapeutic measure is the alleviation of symptoms: if this is not effected, the patient is lost: oral agents have been most helpful in that their use often reduces diabetic symptoms dramatically, and at once the patient feels that something is being done which is helping him.

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I would now like to summarise in full,results of treatment of diabetes in the present series of cases:-

(1). Complete success with the oral anti-diabetic agents is completely subservient to proper dietary measures.

(2). As in the Zulu diabetic, carbohydrate restriction is difficult, then the use of these drugs cannot be said to enjoy complete success.

(3). However, in their elimination of symptoms in a large number of cases, their extreme value is undoubted.

(4). The sulphonylureas tend to increase weights of non-white diabetics disproportionately: thus they should be started in small doses( for instance, 250 mgms Tolbutamide twice daily), and the patient repeatedly urged to cut his carbohydrate intake.

(5). Larger doses may cause such dramatic improvement, and this may lead to a slackening of attempts to diet.

(6). Smaller doses may leave the patient with mild symptoms of his diabetes, such as (improved) nocturia, and this reminds him of the necessity for permanent food restriction.

(7). Insulin was, and is, always popular, as the dose can be pushed up to cover dietary restrictions, and the weight tends to rise rapidly.

(8). The biguanides are invaluable, as they lessen appetite, and thus help with carbohydrate restriction.

(9). In the Zulu patient, in contradistinction to my experiences in Natal Indian and European diabetics, both the sulphonylureas and the biguanides are strikingly free of side-effects.

(10). Very little benefit obtains with the use of the combined sulphonylurea-biguanide combination. Indeed, on clinical grounds, patients who are not dependent upon insulin, appear to be divided into those that respond to sulphonylureas alone, or those that respond to biguanides - in a fuller series, the latter being in the great majority.

(11). Combinations of insulin and sulphonylureas is useless; biguanides

SECTION IV(B) - 9 (continued)

have not been shown to improve control in patients on insulin.
(12). There is seldom any need for any sulphonylurea stronger than Tolbuatmide(Chlorpropamide, acetohexamide or tolazamide). In all cases who really try and restrict carbohydrate, tolbutamide, often in the small initial dosage that I have suggested above, is adequate.
(13). I have never encountered "secondary failure" in Zulu diabetics to sulphonylureas that could not be accounted for by dietary failure.
(14). The oral anti-diabetic agents have been a great boon in the treatment of our type of Diabetic patient in a clinic chiefly concerned with Non-white people in Durban - excldung the upper economic classes.

SECTION IV(B) - 10. THE PREVENTION OF DIABETES.

I feel confident, that in our circumstances certainly, that the principal cause of diabetic emergence in the Zulu people is a disproportionate intake of refined carbohydrate by the urban dwellers, on the grounds of our own dietary comparisons, and the study of epidemiological studies. Thus, as stated above, we have taken particular pains to see that all patients are given our propaganda sheet(p.107) where I have set out what I believe to be the principal causes of diabetes, and factors which aggravate the already-established disease. In this sheet, I have tried to sugfest that diabetes may conceivably be avoidable - whether this is true or not, and to say, that even though we lay stress on the familial connotation of the disease, that there are possibilities if relations of patients avoid obesity, and try and replace highly-refined foods by means of other less refined substances(such as the foods making up the diets of the rural dwellers) that the disease may be avoided. It is difficult to judge effects of this measure except to say, that asking the orderlies to enquire into this, it would appear that our advice has taken on in a most gratifying way. When one considers the enormous amounts of money that are voted annually to the laboratory investigation of diabetes, to the treatment of diabetes, and to the correction of its concomitants or complications, such as(more recently) cardiac transplantation, one cannot help thinking that a mere moeity of these huge sums of money might have an enormous effect possibly in combatting the emergence of diabetes, which is now assuming such a scale in advancing and advanced communities, that I have used the term "The Diabetes Explosion" to describe it. One wonders if such advice might not also be fruitful in countries like Britian or America.

## PART V - PATHOLOGY - CLINICAL AND MORBID.

### SECTION V - 1 - BLOOD SUGAR STUDIES.

A large number of random blood glucose studies were done as a part of the special blood studies reported on page 141. The results of these studies were summarised thus "The blood glucose levels could not be correlated with age, duration of diabetes, or any of the other parameters measured. Because of the varying control in the patients attending this Clinic, the blood glucose values are not reported". (Hathorn, M.K.S., Gillman, T. and Campbell, G.D. 1961. Lancet, i, 1315).

As stated on page 122, I have always been content to regulate the patient's diabetes by his clinical and symptomatic state than by blood sugar. This attitude was mediated by the above observations, and by my earnest endeavour to try and spare the overworked Hospital Laboratory further work. I believe that for all practical purposes in routine work and for the correction of diabetic ketosis and coma, that the Ames Dextrostix (for rapid estimation of blood sugars up to 250 mgms %) is eminently satisfactory in the control of diabetes. Unfortunately glucose oxidase is a fickle substance and goes off very quickly, even in temperate climates, where humidity is high and temperatures high, if one opens the bottle even infrequently, these valuable strips go off very quickly, and I have made an approach to the makers to produce each strip in a metal foil cover. However, even under this apparently favourable circumstance, these strips will still go off quite quickly, and should be purchased only in small quantities in warm places.

### SECTION V - 2 SPECIAL BLOOD STUDIES.

It was at the conclusion of the first year's qualifying follow-up of the patients in this series, having examined all of them carefully, that I first became aware of the enormous discrepancy between the incidence of vascular disease, and of objective neuropathy, in the Natal Indian and the Zulu diabetics. I included objective neuropathy in this consideration because many believe that this syndrome may be due to disease of the vasa nervorum. (Fagerberg, S.E. (1959) Acta Med. Scand. 164, Suppl. 164)

SECTION V - 2 (continued)

Being anxious to try and discover if there might be any significant differences in blood components between these two diabetic populations, one with frequent and the other with uncommon vascular disease, I asked Dr. M. Hathorn and Prof. T. Gillman of the Department of Physiology of the University of Natal (as these tests could not be done in the routine Hospital Laboratory) if they would be good enough to estimate on my behalf certain components in the bloods of groups of patients (a) from the present series of Zulu diabetics and (b) from comparable groups of Natal Indian diabetics. Certain striking differences were found and these were fully reported (Hathorn, M.K.S., Gillman, T. and Campbell, G.D. (1961) Lancet, i, 1314).

Here I wish to record results of the studies done on patients from the present series: in all, a total of 34 males and 48 females from all age groups were studied.

Mean plasma fibrin, serum-micoproteins, total lipids, and Cholesterol in Zulu Diabetics and Controls.

| GROUP | PLASMA-FIBRIN | S.M.P. | SERUM-TOTAL LIPIDS | SERUM-CHOLESTEROL |
|---|---|---|---|---|
| Control (M | 323+18(36) | 86.5+4.4(26) | 749+48(36) | 215+6.3(36) |
| (F | 346+16(24) | 87.3+4.0(22) | 670+47(24) | 213+9.6(24) |
| African Diabetic (M | 436+27(27) | 87.9+1.6(28) | 807+69(34) | 207+8.9(24) |
| (F | 463+21(40) | 99.8+5.4(42) | 836+30(48) | 230+11.0(29) |

(E.L.T.) EUGLOBULIN LYSIS TIME OF DIABETIC SUBJECTS.

| RACE | E. L. T. (NO. OF PATIENTS) | | |
|---|---|---|---|
| | 360min.or less | 361 min.or longer | Total. |
| African Male | 20 | 7 | 27 |
| Female | 24 | 9 | 33 |
| TOTAL | 44 | 16 | 60 |

SECTION V - 2 (continued)

Fibrinoyltic activity is strikingly less in Indians than in Zulu diabetics. Summary of results :-

(A) Plasma fibrin: There were no significant sex differences in mean plasma fibrin levels between male and females in the control or diabetic groups. The mean fibrin levels in Zulu male and Zulu female diabetics were higher than in control groups ($p. < .001$).

(B) Serum Mucoproteins: Any differences between the groups did not reach the 5% level of significance.

(C) Serum Total Lipids: There were no significant differences between the various groups.

(D) Serum Cholesterol: No significant differences were found.

(E) Relationship between Total Lipids, Lysis Time and Cholesterol Levels: Unlike the Natal Indian patients, there were no specific relationships found between these blood components.

(F) Plasma Fibrinolytic Activity: There was no significant difference between male and female Zulu diabetics. The mean E.L.T. for African diabetics was longer than that of 22 non-pregnant and 22 three-twenty one days postpartum Zulu women - something that might simply be explained on the age of the non-diabetic women which was less than that of the diabetics.

## SECTION V - 3 LIVER DISEASE IN THE ZULU DIABETIC.

In 1959, in the King Edward VIII Hospital, Dr. N. McE. Lamont, with Prof. T. Gillman and Dr. M.K.S. Hathorn, did a liver biopsy survey on 400 unselected male ward patients who were not suffering from diabetes. (Gillman, T., Hathorn, M.K.S. and Lamont, N.McE. (1961) - Extrait De Acta Union Internationale Contre le Cancer (Belgium). Vol XVII, Nos. 5 - 6, Chapter 5, 1.)
(Gillman, T., Hathorn, M.K.S., and Lamont, N. McE. (1958). S.Afr. J. Med. Sci., 23, 187) . The overall incidence of severe hepatic siderosis was over 70% At my request they did liver biopsies on 14 male and 2 female diabetics from the present series. In these 16 diabetics, only 2 were found to have iron in their livers, and in one of these it was described as "minimal". This is interesting

SECTION V - 3 (continued)

and fits in well with the fact that the Zulu diabetic is not, on the whole, a heavy beer drinker, and the dictum that in these people diabetes and malnutrition are mutually exclusive. (Page 42 - comparison of diets of diabetics and siderotics).

Clinically the liver was enlarged in only 5 of the 133 at first visit and in 4 this was considered to be due to heart failure. Earlier we were surprised that, as in haemochromatosis, heavy iron deposition in the body should not have resulted in more diabetics in the siderotic Zulu, especially as in the siderotic state, frequent concomitants are testicular atrophy and gynaecomastia and hepatic siderosis. (Higginson, J., Gerritson, T., and Walker, A.R.P. (1953) Am J Path, 29, 779) also showed at that time that in only 7 of 32 heavily siderotic patients did the pancreatic acinar and islet cells contain pigment granules. He noted diabetes to be rare in the Bantu siderotic. This subject has been the basis of an elegant study in Johannesburg (Seftel, H.C., Keeley, K.J. and Walker, A.R.P. (1963). S.Afr. Med. J., 37, 48,1213.)

SECTION V - 4: POST-MORTEM STUDIES.

Six patients were known to have died (Cases Nos. 46, 66, 111, 142, 147, 150).

Case No. 111 died in the Chest Hospital, and a report stated that post-mortem showed widespread acute tuberculosis of the lungs.

Four patients were submitted to post-mortem in our own Hospital :-

Case No. 66: Was found to have a severe bronchopneumonia (see p.74 )

Case No.142: Died in diabetic coma. Nothing abnormal found at P.M. (p. 95 )

Case No.150: Died in irreversible congestive cardiac failure. (p. 66 ) Significant findings were "cardiomyopathy". If this condition is a nutritional disorder, then this case and Case 131 (p. 64 ) - still alive - are interesting as in the general rule, diabetes and nutritional disorder are mutually exclusive in the Zulu - with the exception of the siderotic diabetic (Seftel, H.C., Keeley, K.J., and Walker, A.R.P. (1963). 48, 37, 1214). There was no post-mortem on Case 46, who died in diabetic coma.

## PART VI COMPARISON WITH DIABETICS FROM OTHER RACIAL GROUPS.

### SECTION VI - 1 THE EUROPEANS

Because of widely varying social and dietary considerations, it is not easy to compare the Zulu with the European diabetic. In many clinics it is well-known that a disproportionate number of European patients -especially women - cling to the use of insulin even though they are overweight, because with its use, patients can eat as much as they please, and "cover" extra carbohydrate with extra insulin. Further there are many European diabetics, in whom correct dietary restriction would obviate the need for treatment with the oral anti-diabetic agents. This applies of course to the Zulu patients - the main difference being that the European won't diet whereas, the Zulu can't diet. By and large there are many similarities between the Zulu and the European diabetic, as in both groups there is a substantial number of young patients who are truly dependent upon insulin, and without it lapse speedily into ketosis and coma - as opposed to the Indians(see below). It is probable that the incidence of diabetes is higher in the European than in the Zulu, though in true rural dwellers living on unrefined foods, the incidence in both groups is probably equally low(p. 24).

### SECTION VI - 2 THE NATAL INDIAN.

The chief differences between the Natal Indian and the Zulu diabetic are:-

(a). Diabetes is about 4 times as common in the Natal Indian.

(b). There are far more young diabetics amongst the Natal Indians who are not dependent on insluin, and who are well controlled by oral therapy and carbohydrate restriction.

(c). Vascular complications or concomitants, are far more common in the Natal Indian diabetic than in the Zulu diabetic, where diabetes is apparently of similar duration.(Hathorn, M.K.S., Gillman, T., and Campbell, G.D.(1961). Lancet, i, 1314).

(d). The Zulu is far better at trying to cooperate with measures laid down for the treatment of diabetes.

(e). There are certain striking differences in blood components of the Zulu diabetic(vascular disease not very common) and the Natal Indian (vascular disease common). (Hathorn, M.K.S., Gillman,T., and Campbell, G.D. (1961). Lancet, i, 1314).

## PART VII FINAL SUMMARY OF THE THESIS.

This thesis describes a clinical study, over 9 years, of diabetics amongst a group of people - the Zulus - in whom up until 15 years ago diabetes was considered to be a most uncommon disorder.

Possible factors contributing to the emergence of diabetes in urban as opposed to rural dwellers have been considered, and the overwhelming body of evidence points to the disproportionate ingestion of refined carbohydrates as being the major cause for this.

A number of patients were taken(133) on the grounds of having attended the Clinic for a full year, and these were followed up for up to 9 years, and standards of follow-up(regularity and total length) have been carefully analysed.

The incidence of the complications - or concomitants - of diabetes was carefully assessed at the first visit of each patient in the series, and each complication was discussed under an appropriate heading. These clinical features were discussed under the headings of (a)"Vascular" - pertaining to the blood vessels: (b) "Infective" - that is to say the incidence of all infective disorders that were noted in the period of follow-up, and (c) "metabolic", referring to those conditions probably chiefly due to the humoral changes in body fluids in diabetes, and including other conditions that do not come into the "vascular" or "infective" sub-divisions. In every instance, attempts were made to equate the presence of these conditions with the known duration of diabetes.

All patients were examined every 6 months, unless symptoms and signs suggested that more frequent examination was necessary. Where patients developed diabetic complications or concomitants after their initial examination, these are listed in detail under the appropriate section, in paragraphs labelled "follow-up cases". In view of the variable duration of follow-up of many cases, no attempt was made to describe the cumulative incidences of the various complications. Thus the figures of incidence quoted refer to the incidence of these conditions that were noted within 1 year in the series of diabetics, and not those that developed subsequently. As the duration of diabetes when patients were first seen varied between 0 and 12 years, it appeared reasonable to study incidence of complications over a short period as indicating the "true" incidence in any respresentative diabetic group.

Effects of the various forms of treatment over the whole follow-up have been studied, and the advantages of the oral anti-diabetic agents stressed.

PART VII(continued)

I have also set out criteria of control which I believe to be of practical value under our present circumstances.

Certain complicated blood studies were done, in an attempt to elucidate on a clinical pathology basis, the remarkable discrepancy between the incidence of vascular disease in the Zulu and in the Natal Indian diabetics. Results pertaining to the Zulu diabetics have been included in the thesis. Ample reference has been made to factors that militate against good control and satisfactory clinic attendance.

Suggestions have been made as how best to combat the extreme difficulties that beset anyone contemplating starting a Diabetic Clinic for semi-literate people, in whom insight into the disease may be lacking.

A special section has been included which contains that part of the Book- "Handbook to Aid in the Treatment of Zulu Patients"(Campbell, G.D. and Lugg, H.C.(1961). Natal University Press), which pertains specially to (a) The Diet Sheet:(b)Phrases for usage in the Diabetic Clinic and (c) Phrases with which to instruct in drug dosage - as a guide to anyone wishing to start a Diabetic Clinic for Non-English-speaking people, who can thus adapt the English half of the pages for whatever other language they may wish to use.

Again, I have stressed the necessity for doctors working amongst semi-literate patients to identify themselves as much as they can with their patients(Campbell, G.D.(1963). E. Afr. med. J., 5, 272), otherwise only a very loose doctor-patient relationship can exist, which is not conducive to proper treatment or patient confidence.

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APPENDIX ONE.

LIST OF PATIENTS WITH CLINIC REFERENCE NUMBERS

Summary of Breakdown by Sexes in Diabetic Groups

| | | | | |
|---|---|---|---|---|
| 133 Patients: | A. Insulin-dependent | | | |
| | | Male | 16 | |
| | | Female | 16 | 32 |
| | B. Middle-aged | | | |
| | | Male | 32 | |
| | | Female | 56 | 88 |
| | C. Senile | Male | 5 | |
| | | Female | 8 | 13 |
| | | TOTAL | | 133 |

## A - INSULIN-DEPENDANT.

| MALES: (16) | NAME |
|---|---|
| A/3 | Jothan SHABANGU |
| A/30 | John MTSHALI |
| A/44 | Christopher KUMALO |
| A/63 | Alpheus NDLOVU |
| A/98 | Alfred KUZWAYO |
| A/99 | John NGCOBESE |
| A/110 | Kosi NYAWO |
| A/112 | Bernard MBAMBO |
| A/136 | Jiza GWALA |
| A/145 | George MDIMA |
| A/147 | Elsie RAMBA |
| A/148 | Jacob CONCO |
| A/150 | Elias KUMALO |
| A/161 | Nathan MNGOMA |
| A/163 | Mbingelelwa KUZWAYO |
| A/177 | Zibonela MTEBENI |

| FEMALES: (16) | |
|---|---|
| A/4 | Constance NTOMBELA |
| A/6 | Joanna MDLADLOSE |
| A/14 | Constance NDABA |
| A/19 | Constance MAKHANYA |
| A/25 | Theresa MABTHA |
| A/27 | Jocelyn BHOPHELA |
| A/42 | Elizabeth SIBISI |
| A/49 | Eldis KUMALO |
| A/94 | Clerwina KHLUNGWANE |
| A/100 | Maggie NGONYAMA |
| A/118 | Melonia NGCOBO |
| A/126 | Maggie SIKAKANE |
| A/141 | Ritta NDHLOVU |
| A/142 | Agnes DLOMO |
| A/170 | Ntombizinonyaka NGCOBO |
| A/174 | Busisizwe KUMALO |

## B: MIDDLE AGED.

MALES: (32)

| | NAME |
|---|---|
| A/9 | Fred NGCOBO |
| A/20 | Job MAKHANYA |
| A/21 | Dumangese DHLAMINI |
| A/33 | Jeconiah MTHEMBU |
| A/35 | Charlie MNGADI |
| A/37 | George SHEZI |
| A/44 | Christopher KUMALO |
| A/50 | Solomon MBATHA |
| A/52 | Paulus SONDEZI |
| A/57 | Tom NGCABASHE |
| A/72 | Almon MAKATINI |
| A/75 | Sofa MLAMBO |
| A/80 | Phineas NTANZI |
| A/85 | Mkuzwa CELE |
| A/87 | Mdabula HLOPE |
| A/97 | Maurice MKWANAZI |
| A/105 | Simon MENDU |
| A/107 | Philip DHLAMINI |
| A/114 | Alpheus NDHLOVU |
| A/119 | Christopher NXUMALO |
| A/125 | Alfred MHLONGO |
| A/127 | Josiah HLOPE |
| A/130 | Gilbert KHANYILE |
| A/131 | Dittimus DHLAMINI |
| A/154 | Richard MDLULI |
| A/157 | William GUMEDE |
| A/168 | Jan MADLALA |
| A/169 | Levi RADEBE |
| A/184 | Amon ZAKWE |
| A/185 | Tom NGCOBO |

B: MIDDLE AGED.

| FEMALES: (56) | NAME |
|---|---|
| A/5 | Elsie SIBISI |
| A/7 | Rhoda MTEMBU |
| A/8 | Beauty MHLONGO |
| A/13 | Josephine UMBULU |
| A/16 | Juliet VILIKAZI |
| A/24 | Josephine MAKANYA |
| A/29 | Agnes KWEYAMA |
| A/38 | Gladys MSOMI |
| A/40 | Fanny MSANE |
| A/41 | Flora DHLAMINI |
| A/43 | Adelina MALINGA |
| A/45 | Mahlangoti DHLAMINI |
| A/46 | Alzina MAPHAPHALALA |
| A/48 | Norah NGWENYA |
| A/53 | Bessie MAVUNDLA |
| A/61 | Lucy NHLAPO |
| A/62 | Violet DAMISA |
| A/65 | Roseline DHLOVU |
| A/66 | Lena SOSIBO |
| A/67 | Nani DUBE |
| A/71 | Taureen NDHLOVU |
| A/73 | Jessie KHANYILE |
| A/77 | Lena KUBEKA |
| A/82 | Rose MOSERY |
| A/84 | Theresa BHENGU |
| A/86 | Alosia DHLAMINI |
| A/91 | Eslina NGCOBO |
| A/93 | Francis MKIZE |
| A/95 | Seraphina MAPUMULO |
| A/96 | Catherine GUMEDE |
| A/101 | Gertrude KUMALO |
| A/103 | Annie MHLONGO |
| A/104 | Evelyn MZIMELA |

B: MIDDLE AGED (continued)

FEMALES: (56)

| | NAME |
|---|---|
| A/108 | Alzina SHALIZAN |
| A/120 | Rose DHLAMINI |
| A/121 | Martha NGUBENI |
| A/123 | Elizabeth MALETI |
| A/132 | Judith MZAMO |
| A/134 | Lesiah MADI |
| A/137 | Maggie NSUZA |
| A/139 | Sellina MNSMZANE |
| A/140 | Kate MAPUMULO |
| A/143 | Norah MKIZE |
| A/146 | Gertrude NGUBANE |
| A/151 | Violet MKWANAZI |
| A/152 | Ivy FRANCIS |
| A/153 | Elsie MLADLOSE |
| A/156 | Ivy NKIZE |
| A/159 | Gertrude MTETWA |
| A/160 | Salvina MDLAZI |
| A/162 | Flora KUBEKA |
| A/164 | Elverd DLAMINI |
| A/166 | Astina DUMA |
| A/167 | Mina MTEMBU |
| A/175 | Albertina NGCOBO |
| A/180 | Ivy MKIZE |

## C: SENILE

**MALES: (5)**

| | NAME |
|---|---|
| A/122 | Mdewa SIKONDE |
| A/133 | Samuel SIMELANE |
| A/144 | Mpiyonke XULU |
| A/149 | Basa DZAMIBE |
| A/165 | Goli MVUNDLE |

**FEMALES: (8)**

| | |
|---|---|
| A/2 | Elizabeth KUMALO |
| A/34 | Margaret MHLONGO |
| A/90 | Emmah KAMBULE |
| A/111 | Linah NTULI |
| A/115 | Josephine MASEKU |
| A/116 | Mamane GUMEDE |
| A/128 | Jemima KAMBULE |
| A/129 | Lizzie MAZUKU |

APPENDIX TWO - 1

A DIET SHEET FOR OBESE AND DIABETIC PATIENTS.

"Handbook to Aid in the Treatment of Zulu Patients" - Campbell G.D., and Lugg H.C., (Page 50) 1961 (University of Natal Press)

Advice and Admonitions:-

| English | Zulu. |
|---|---|
| -- To be excessively fat, is to court death. | Ukuzimuka Kakhulu ukafa. |
| Your gross obesity disturbs one. | Ukukhuluphala kwako kuyasabisa. |
| You are as fat as a pig! (as a hippo!) | Ucebise kwengulube. (Kwenvubu). |
| You are not following the diet prescribed. (Lit. You are not eating the doctor's food). | Awukudli Ukudla ka dotela. |
| Food is the cause of fat (i.e. obesity). | Ukudla Kubanga amafuta or Ukudla kubanga ukuzimuka. |
| Lose weight, and you will get better. (Lit. Cut down on your fat....) | Phungula isisindo. Phungula amafuta, khona uzosinda. |
| Fat people die before their time. | Abazimukile bafa besebasha. |
| FAT PEOPLE SHOULD STRICTLY AVOID THE FOLLOWING FOODS:- | ABAZIMUKE KAKHULU ABANGAKU LOKOTHI UKUDLA OKUNJENGA LOKU:- |
| SUGAR - or any sweetened foods such as jams, tinned fruit and lemonade. | USHUKELA - nokudla okunandisiwe ngo shukela, njengo jam, amafruit ethini, nolemonade. |
| BREAD - or eat only three slices daily. | ISINKWA - Idl'amacezwana amathathu ngelanga kuphela. |
| POTATOES - Eat as little phuthu, samp and mealie rice as possible. | AMAZAMBANE - Idla kancane uphutu, isitambhu, ne mealie rice. |
| BEER - Take only two cups daily. | UTSHWALA - Phuz'izinkomishi ezimbili ngelanga kuphela. |
| BUT YOU (FAT PERSON):- | KODWA WENA OKUKHULUPHELA:- |
| Eat all the green vegetables you can. | Idla kakhulu imfino yonke. |
| Eat a lot of fruit. | Idla kakhulu amafruit. |
| Eat meat, fish, tripe, chicken, eggs and whale meat. | Idla inyama, inhlanzi, usu, inyama yenkuku, amaqanda, neyama yomkhomo. |

| English | Zulu. |
|---|---|
| Check your appetite with extra tea. | Khuz'iphange ngetiye. |
| SACCHARINE - Eat this, and not sugar | USACCHARINE - (Amapilisi ka shukela). Idla wona. Ayi shukela. |
| EXPENSE - As this diet is expensive, don't buy tobacco and beer. | Ngoba lokudla kuyabiza, ungatengi ugwayi notshwala. |

APPENDIX TWO - 2

ZULU PHRASES FOR USE IN THE DIABETIC CLINIC.

"Handbook to Aid in the Treatment of Zulu Patients"

Campbell G.D., and Lugg H.C.

1961 (University of Natal Press)
(Page 47)

| English | Zulu |
|---|---|
| Diabetes (the illness of sugar.) | Isifo sika shukela. |
| Insulin (the substance injected in the illness of sugar.) | Umjovo wesifo sika shukela u-Insulin. |
| SYMPTOMS - How many times do you pass water at night? | Uthunda (uchama) kangaki ebusuku? |
| Does it burn when you urinate? | Kuyashisa uma uchama? |
| Any boils or sores? | Amathumba akhona, noma izilonda? |
| Are you losing weight? | Lomjovo uyagulisa? |
| REACTIONS - Does this injection upset you? | Lomjovo uyagulisa? |
| Have you been sweating or shivering? | Ayajuluka noma uyagodola? |
| DIETING (See page 50, Diet Sheet). | |
| Are you dieting? (Lit. Do you reduce your food?) | Ukudla uyakuphungula? |
| You are too fat! | Uzimuke kabi! |

| English | Zulu |
|---|---|
| You're eating too much.<br>Eat three equal sized (Lit. small) meals daily. | Udla kakhulu wena.<br>Udla kancane kathathu ngelanga. |
| DON'T EAT ! !<br>Sugar, bread, potatoes, sweets, lemonade and beans. | UNGADLI ! !<br>Ushukela, isinkwa, amazambane, oswidi, ulemonade, nobontshisi. |
| URINE TESTING - Have you been testing your urine regularly? | Uyauhola umchamo njalo? |
| What colour has it been mostly?<br>Brick?<br>Orange?<br>Yellow?<br>Green?<br>Blue? | Kumbala muni?<br>Kubomvana?<br>Kumbomvu ngokumpofu?<br>Mpofu?<br>Luhlaza?<br>Luhlaza ngokumnyama? |
| CHEST X-RAYS - When was your chest last X-rayed? | Isifuba sathatwe nini isithombe? |
| I want one now.<br>Let it (the X-ray) be taken yearly. | Ngifuna esinye manje.<br>Asithatwe njalo ngonyaka (isithombe). |
| REPLENISHMENT OF DRUGS<br>Do you want (any more)....<br>Insulin? | Usafuna .....<br>Umjovo wesifo sika shukela?<br>u-Insulin? |
| Ether?<br>Meths?<br>Cotton Wool?<br>Benedict's? (Lit. Green medicine to test urine). | Umuthi wokuhlikihla?<br>Ispiriti samalambu?<br>Uvolo?<br>Umuthi oluhlaza (wokuhlola umchamo)? (u-Benedicts.) |
| INJECTION TECHNIQUE -<br>Show me your syringe.<br>Draw it up to this mark.<br>Inject yourself always at 7 a.m.<br>Inject yourself in different places; in the legs, the arms, the abdomen. | Ngibonise inglazi yomjovo.<br>Donsa uzufike kulomaka.<br>Zijove ngo 7 ekuseni.<br>Jova izingalo, imilenze, nomazimba. |
| If you have a reaction (Lit. If the injection makes you sick), lower the dose by one mark. | Uma uguliswa umjovo, ehlisa umaka. |

| English | Zulu |
|---|---|
| HOW TO TEST YOUR URINE - To test your urine, proceed as follows :- | Ukuhlola umchami kuhamba kanje :- |
| Fill the test tube to the depth of your thumb. | Gewalisa umtshumana wenglazi ngomuthi oluhlaza, kuze kuli-ngane nesithupa (Demonstrate.) |
| Add eight drops of urine from your dropper. | Tela amaconsi awu 8 omchamo. |
| Boil it thus. | Bilisa kanje. |
| Watch that it doesn't froth up suddenly. | Basobha kunga phuphumi. |
| Boil for one minute. | Bilisa iminthi. |
| The colour shows the amount of sugar. | Umbalo Ukhombisa ubuningi buka shukela. |
| There is a lot if it is brick. (Lit. red.) | Mningi uma ubomvu. |
| There is a little if it is yellow. | Uningana uma mpofu. |
| There is none if it is blue. (Lit. green) | Ize uma uhlaza. |
| ADVICE:- If there is a lot of sugar, increase the do e by one mark-thus. | Uma ushukela umningi, enyusa umaka - kanje. |
| Always come with your empty bottles and syringe. | Buya njalo namabodlela nesirinji. |
| Is that quite clear? (Lit. Have you heard?). | Uzwile? |

APPENDIX TWO - 3.

## INSTRUCTIONS ABOUT TAKING DRUGS

"Handbook to Aid in the Treatment of Zulu Patients"

Campbell G.D., and Lugg H.C.,

1961 (University of Natal Press)
(Page 36)

In this key, instructions about taking tablets, medicines and powders are laid out; the sentence is divided into three parts:-

Section A: The Introductory Order.
Section B: The descriptions of the drug, and its dose.
Section C: The frequency with which the drug is to be taken.

The sentence is made up by moving from Section A to B and then to C, taking from each section the particular phrase that is wanted.

SECTION A - THE INTRODUCTORY ORDER

| English | Zulu |
|---|---|
| I want you to take..... | Ngifuna ukuba udle ..... |

(Now go to Section B - B1 for tablets, and B2 for mixtures and powders.)

SECTION B .

B1 - TABLETS

| | |
|---|---|
| .... of the pills, | ....Amapilisi, |
| ..... brown | ..... ansundu |
| black | amnyama |
| white | amhlope |
| green | aluhlaza |
| red, | abomvu, |
| .... one | .... Libe linye |
| two | Abe mabili |
| three | Abe mathatu |

(Now go to Section C - over)

B2 - POWDERS AND MIXTURES

| | |
|---|---|
| .... of the mixture | .... Umuthi Webodlela |
| of the powder, | Umuthi Oyimphuphu, |
| .... one tablespoon | .... Itablespoon |
| two tablespoons | Amatablespoon amabili |
| three tablespoons | Amatablespoon amathathu. |

..

| English | Zulu |
|---|---|
| .... one teaspoon | .... Iteaspoon |
| two teaspoons | Amateaspoon amabili |
| three teaspoons | Amateaspoon amathathu. |

SECTION C - FREQUENCY

| | |
|---|---|
| .... Once daily | .... Kanye ngelanga |
| Twice daily | Kabili ngelanga |
| Three times daily.. | Kathathu ngelanga. |
| .... After meals | .... Ngemuva khwo kudla |
| Before meals | Ungakadli .... |
| .... At night | .... Kusihlwa |
| .... When the pain comes | .... Uma uzwa kubuhlungu. |